VOLUME 18 NUMBER 3

AMERICAN JOURNAL

OF

DISEASES OF CHILDREN

EDITORIAL BOARD

[illegible]

L. EMMETT HOLT, New York City DAVID H. COMEY, Ann Arbor, Mich.

JOHN LOVETT MORSE, Boston EDWIN E. GRAHAM, Philadelphia

JOHN HOWLAND, Baltimore

SEPTEMBER, 1919

PUBLISHED MONTHLY BY THE AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ENTERED AS SECOND-CLASS MATTER, MAY [illegible]

CONTENTS OF PREVIOUS NUMBERS

MARCH, 1919. NUMBER 3.

Epidemic Influenza in Infants. Martha Wollstein, M.D., and Alton Goldbloom, M.D., New York.

The Relation of Prenatal Nativity to the Infant Mortality of New York State. P. E. Eastman, Albany, N. Y.

Report of an Unusual Case of Aplastic Anemia. Lawrence Weld Smith, A.B., Boston.

A Simple Method of Modifying the Fat Content of Milk for Infant Feeding. Amy L. Daniels and Honora English, Madison, Wis.

Current Pediatric Literature.

APRIL, 1919. NUMBER 4.

Scurvy VIII. Factors Affecting the Antiscorbutic Value of Foods. A. F. Hess, M.D., and L. J. Unger, M.D., New York.

A Study of the Fat Metabolism of Infants and Young Children. I. Fat in the Stools of Breast Fed Infants. L. E. Holt, M.D., A. M. Courtney and H. L. Fales, New York.

A Nutrition Clinic in a Public School. W. R. P. Emerson, M.D., Boston.

Bovine Tuberculosis in Children. R. S. Austin, M.D., Chicago.

Studies in the Nephritis of Children. Clinical Considerations of Classification, Etiology, Prognosis and Treatment. L. W. Hill, M.D., Boston.

Current Pediatric Literature.

MAY, 1919. NUMBER 5.

The Cystoscopic Study of Urologic Conditions in Children. Frank Hinman, A.B., M.D., F.A.C.S., San Francisco.

Studies of the Mammary Gland. V. Effects of Inanition on the Developing Mammary Glands in Male and Female Albino Rats from Birth to Ten Weeks of Age. J. A. Myers, Ph.D., Minneapolis.

The Measurements of 250 Full-Term New-Born Infants. Rood Taylor, M.D., ScD., Minneapolis.

Recovery of Normal Weight in the Various Organs of Albino Rats on Refeeding After Underfeeding from Birth for Various Periods. C. M. Jackson, M.S., M.D., and C. A. Stewart, Ph.D., Minneapolis.

Progress in Pediatrics: Review of Recent Literature on the New-Born. N. O. Pearce, M.D., Minneapolis.

Current Pediatric Literature.

JUNE, 1919. NUMBER 6.

Influenzal Croup. Joseph C. Regan, M.D., and Catherine Regan, M.D., Brooklyn.

Fat Metabolism of Infants and Young Children. II. Fat in the Stools of Infants Fed on Modifications of Cow's Milk. L. Emmett Holt, M.D., Angelia M. Courtney and Helen L. Fales, New York.

Clinical Department: Infantile Scurvy Associated with Hereditary Syphilis. Report on a Breast Fed Infant with Multiple Fractures. R. M. Greenthal, M.D., Ann Arbor, Mich.

Some Graphs and Tables Illustrating the Growth of the Human Stomach. Richard E. Scammon, Ph.D., Minneapolis.

Progress in Pediatrics: Current Pediatric Literature.

JULY, 1919. NUMBER 1.

The Precipitin Reaction in Infant Stools. Ludvig Hektoen, M.D., and Clifford G. Grulee, M.D., Chicago.

Studies of the Mammary Gland. VI. The Development of the Mammary Gland from Its Earliest Appearance Until the Period of Pregnancy. J. A. Myers, M.S., Ph.D., Minneapolis.

Bacillus Tuberculosis in the Tonsils of Children Clinically Non-Tuberculous. R. S. Austin, M.D., Chicago.

Clothing of Infants in Summer and Winter. L. W. Sauer, M.D., Chicago.

A New Stain for the Diphtheria Bacillus and a Consideration of the Principles Involved in the Staining of this Organism. Roy M. Greenthal, M.D., Ann Arbor, Mich.

The Antiscorbutic Property of Fruits. I. An Experimental Study of Dried Orange Juice. Maurice H. Givens and Harry B. McClugage, Rochester, N. Y.

The Acidotic State of Normal New-Borns with Special Reference to the Alveolar CO_2 Tension, Alkali Tolerance and Acetonuria. Max Seham, M.D., Minneapolis.

Progress in Pediatrics:
Review of Recent Literature on the New-Born. N. O. Pearce, M.D., Minneapolis.

Current Medical Literature.

AUGUST, 1919. NUMBER 2.

A Study of the Relationship of Convulsions in Infancy and Childhood to Epilepsy. John Lovett Morse, M.D., Boston.

Postinfluenzal Encephalitis. Henry Heiman, M.D., New York.

The Constipating Qualities of Orange Juice. H. J. Gerstenberger, M.D., and W. M. Champion, A.B., M.D., Cleveland.

Fat Embolism of Infants and Young Children. III. Fat in the Stools of Children on a Mixed Diet. L. Emmett Holt, M.D., Angelia M. Courtney and Helen M. Fales, New York.

A Study of the Lactose, Fat and Protein Content of Women's Milk. W. Denis, Ph.D., and Fritz B. Talbot, M.D. (with the assistance of A. S. Minot), Boston.

The Cause of a "Sporadic" Case of Cerebrospinal Meningitis. Henry L. K. Shaw, M.D., Albany, N. Y.

A Classical Case of Hemophilic Arthritis. May G. Wilson, M.D.

Progress in Pediatrics:
Review of Recent Literature on the New-Born. N. O. Pearce, M.D., Minneapolis.

Current Pediatric Literature.

Book Review.

American Journal of Diseases of Children

Vol. 18 SEPTEMBER, 1919 No. 3

LEUKOCYTE COUNTS DURING INFLUENZA IN INFANTS AND CHILDREN *

J. C. MONTGOMERY, M.D., AND E. C. DUNHAM, M.D.
BALTIMORE

During the epidemic of influenza prevalent in Baltimore in October, 1918, we had the opportunity to observe a series of cases in infants and children. It is our purpose to give a brief summary of the leukocyte counts in these cases, with some observations on their apparent relation to the severity and complicating factors of the disease.

Our material is drawn from cases seen and treated in the Harriet Lane Home and in the Harriet Lane Dispensary. In addition to these, approximately 150 patients were seen on extern visits. In these, however, there was no opportunity for blood studies, and the cases could not be followed carefully, hence they are not included in this report.

Eighty-one patients, in all, were followed in the dispensary and in the hospital. Owing to conditions these were of necessity the more critically ill children, and this must be borne in mind in considering the counts which follow. Of these seventeen, or 20.9 per cent., were infants (under 1 year of age) and sixty-four, or 79.0 per cent. were children.

The incidence of pneumonia and the mortality are shown in Tables 1 and 2.

TABLE 1.—INCIDENCE OF PNEUMONIA

	Number of Cases	Pneumonia	Percentage of Pneumonia
Infants	17	9	52.9
Children	64	26	40.6
Total	81	35	43.2

TABLE 2.—MORTALITY FROM INFLUENZA AND PNEUMONIA

	Number of Cases	Deaths	Mortality, per Cent.
Infants—			
Influenza	8	1	1.25
Pneumonia	9	5	55.5
Children—			
Influenza	38	0	0.0
Pneumonia	26	10	38.46
Total	81	16	19.7

* From the Pediatric Department of the Johns Hopkins University and Harriet Lane Home, Johns Hopkins Hospital, Baltimore.

In Table 3 we have summarized our total leukocyte counts, both in influenza and in cases complicated by pneumonia.

TABLE 3.—Leukocyte Count in All Cases

	No. of Cases	White Blood Cells		
		Average	Maximum	Minimum
From birth to 3 months—				
Influenza	1	11,000		
Pneumonia	0			
From 3 to 6 months—				
Influenza	0			
Pneumonia	1	6,000		
From 6 months to 1 year—				
Influenza	2	13,000	14,000	12,000
Pneumonia	2	11,700	17,400	6,000
From 1 to 5 years—				
Influenza	6	6,745	16,000	4,120
Pneumonia	9	11,471	28,120	3,640
From 5 to 12 years—				
Influenza	5	7,728	11,000	3,800
Pneumonia	4	8,075	17,200	3,840
Total	30			

In reviewing these results, the following points seem to be of interest:

1. There was, in general, an absence of leukocytosis, particularly in the uncomplicated cases.

2. In 14 of the 30 cases in which blood studies were made or roughly, 50 per cent., the leukocyte count on admission was 6,000 or below. The average counts are at about the normal levels. The averages, and the maximum and minimum counts for the different ages are grouped in Table 3.

3. In general, those cases complicated by pneumonia will be seen to have had higher counts than uncomplicated cases. An analysis of the pneumonia patients in relation to mortality is given in Table 4.

TABLE 4.—White Blood Cell Count in Pneumonia

	Average	Maximum	Minimum
Recovered (7)	14,371	28,120	6,000
Died (6)	5,770	9,320	3,640

It will be seen that in all fatal cases the leukocyte counts were under 10,000, and that the average was much lower than in the cases in which recovery ensued. It is also of interest to note that of the pneumonia patients with leukocyte counts less than 10,000, six, or 66.6 per cent., died, while of those having counts above 10,000, none died. In our experience the leukocyte counts showed a tendency to rise during convalescence. To illustrate: One patient with a count of 9,800 at the height of the disease, had a leukocytosis of 16,080 during convalescence.

Special mention should be made of the one fatal case which was thought to be uncomplicated by pneumonia. This child was seen in the dispensary when four days of age. The mother, still bedridden, was definitely suffering from influenza. The father died later from pneumonia. This infant had a temperature of 103.2 F., and a leukocyte count of 11,000, coryza, and marked pharyngeal injection. The patient died at the age of 14 days. Although no clinical evidences of pneumonia were discovered, it cannot be stated definitely that it did not exist.

Differential counts were made in nine cases — three cases of uncomplicated influenza and six cases of influenza and pneumonia. Our results are given in Table 5, to be compared with that of the normal child, between 6 months and 11 years of age.[1] It will be seen that the polymorphonuclear neutrophils are increased about equally in the complicated and uncomplicated cases.

TABLE 5.—DIFFERENTIAL LEUKOCYTE COUNTS

	Influenza without Pneumonia			Influenza with Pneumonia					
	Three Patients, Aged from 7 months to 11 years			Six Patients, Aged from 5 months to 6 years			Normal Children, Aged from 6 months to 11 years		
	Average	Maximum	Minimum	Average	Maximum	Minimum	Average	Maximum	Minimum
Polymorphonuclears	61.6	69	48	63.6	80.0	46	49.4	56.4	42.6
Basophils	0.33	1	0	0.16	1.0	0	0.36	0.73	0.07
Eosinophils	0.0	..	0	0.16	1.0	0	2.4	4.36	0.95
Large mononuclears and transitionals	12.3	19	8	7.8	14.0	3	9.0	12.6	5.7
Small mononuclears	25.6	32	21	27.1	48.0	12	38.0	44.4	30.9

In Table 6 are shown the polymorphonuclear percentages in the six cases of influenza, complicated by pneumonia, in which differential counts were made.

TABLE 6.—POLYMORPHONUCLEAR PERCENTAGES IN INFLUENZA

	Patients Discharged Well		Patients Died	
Polymorphonuclears	1	46%	1	70%
	2	56%	2	72%
			3	80%
			4	58%

CONCLUSIONS

1. The tendency in uncomplicated influenza in infants and children is toward a leukopenia, rather than a leukocytosis.

2. There is a tendency to a slight leukocytosis in complicating pneumonia.

1. Schloss, O.: Arch. Int. Med. **6**:638 (June) 1910.

3. In this series, in all pneumonia cases resulting fatally the leukocyte counts were under 10,000.

4. The prognosis in general is better in pneumonia cases which exhibit a leukocytosis.

5. Differential counts have shown: (a) A tremendous variation in the differential formula, and (b) nothing sufficiently constant to be of clinical aid in diagnosis or prognosis.

FAT METABOLISM OF INFANTS AND YOUNG CHILDREN *

IV. THE DIGESTION OF SOME VEGETABLE FATS BY CHILDREN ON A MIXED DIET

L. EMMETT HOLT, M.D.
ANGELIA M. COURTNEY AND HELEN L. FALES
NEW YORK

During the last few years there has been a steadily increasing use of vegetable fats, especially nut butter, in the diet of children. This has been due chiefly to the scarcity and the greatly increased cost of milk and butter. Recently, vegetable fats have been used extensively both in the wards and the out-patient department of this hospital. It seemed fitting, therefore, that a study on fat metabolism should include observations on children taking certain of these vegetable fats. While the number of cases on which laboratory studies have been made is not large, enough observations are presented to allow some conclusions to be drawn.

It is not intended in this paper to suggest that vegetable fats are equivalent to milk fat or can entirely replace it in the dietary of children. It is well known that such is not the case. It has been shown by Osborne and Mendel[1] and by McCollum[2] and others[3] that milk fat, as well as some other animal fats, contains a constituent necessary for normal growth and maintenance, to which the name "fat-soluble A" has been given. This is often referred to as the fat soluble vitamin. It has been found not to be present in vegetable fats to any considerable extent. Although this substance is present in certain other foods, especially eggs, and to some extent in the vegetative parts of plants, for example, spinach, lettuce, cabbage, it is evident that these articles cannot be given to young children in large amounts. Milk and butter are the foods which most easily provide a supply of the fat soluble vitamin ample for the needs of children, for as yet the minimum quantity of this substance required by the growing child has not been determined.

---

* From the Laboratories of the Rockefeller Institute for Medical Research and the Babies' Hospital, New York.

1. Osborne and Mendel: J. Biol. Chem. **16**:423, 1913; ibid. **17**:401, 1914.
2. McCollum and Davis: J. Biol. Chem. **15**:167, 1913. McCollum, Simonds and Pitz: Am. J. Physiol. **41**:361 (Sept.) 1916.
3. Halliburton and Drummond: J. Physiol. **51**:235 (Sept.) 1917.

The total fat requirement of children, however, undoubtedly exceeds the amount of milk fat which is needed to supply a sufficient quantity of this vitamin. Vegetable fats, if equally digestible, may therefore be substituted to a considerable degree for milk fat in supplying the fat needed in the diet of children.

Langworthy and Holmes[4] have tested the digestibility of vegetable fats in adults and found that, with the exception of cocoa fat, all those tested, including cocoanut oil and corn oil, were digested quite as well as milk fat. Rockwood and Swickes[5] found that dogs utilized corn oil perfectly well. No reports with laboratory findings of the digestibility of vegetable fats in children have come to our notice.

The observations here reported were made on children who received a mixed diet containing varying amounts of vegetable fat in the form either of nut butter or corn oil. For nut butter several of the brands of nut margarine on the market were used. These margarines consist mainly of cocoanut oil and are labeled as free from animal fat. Cocoanut oil consists mainly of the glycerids of palmitic and oleic acid, in this respect resembling milk fat; but it contains a smaller proportion of olein than does milk fat. Cocoanut oil also contains considerable amounts of myristin and laurin. The melting point of nut butter is slightly lower than that of milk butter, so that it is barely solid at room temperature. This nut butter is white and almost tasteless when fresh, but becomes rancid more easily than does milk butter.

Corn oil is composed largely of the glycerids of palmitic and oleic acid, with a higher proportion of olein than is present in milk fat. It is a liquid, somewhat deeper in color than olive oil. When fresh, it is almost tasteless and odorless, but acquires a somewhat unpleasant taste and odor on continued exposure to the air.

In order to investigate the digestibility of corn oil and nut butter, analyses were made of the stools of children taking varying amounts of these fats. The feces were collected, dried and analyzed according to the procedure followed in previous investigations of fat in stools.

The first part of this paper gives the fat percentage and distribution of fat in the stools and the retention of fat of these children, arranged in groups according to the proportion of the total fat in the diet which was vegetable fat. The second part of the paper gives for different children the analyses of the stools and the fat retention with various changes in fat intake, both as to the kind of fat and the proportions of vegetable fat and milk fat, together with the principal clinical symptoms with each change.

---

4. Langworthy and Holmes: U. S. Dept. Agricul. Bull. 505, 1917. Holmes: U. S. Dept. Agricul. Bull. 687, 1918.
5. Rockwood and Swickes: J. A. M. A. **71**:1649 (Nov. 16) 1918.

PART I.

Tables 1 to 7 give the findings in forty-four observations on fourteen children receiving varying proportions of their fat intake in the form of nut butter or corn oil. These children had a general mixed diet. All received milk, usually over one pint daily. In some cases wholly or partially skimmed milk was given in order to reduce the milk fat in the diet. It was necessary, in order to maintain a sufficient intake of salts, especially those of calcium, to use this method of reducing the intake of milk fat instead of diminishing the quantity of milk in the diet. The rest of the food consisted of cereal, bread, vegetables, fruits and usually egg or meat or both.

Tables 1 and 2 give the findings in the instances in which the vegetable fat formed from 20 to 35 per cent. of the total fat intake, Table 1 when the stools were alkaline and Table 2 when they were acid. In the cases in these two tables the vegetable fat was nut butter.

TABLE 1.—FAT IN STOOLS AND RETENTION OF FAT OF CHILDREN RECEIVING FROM 20 TO 35 PER CENT. OF THE TOTAL FAT INTAKE AS NUT BUTTER. STOOLS ALKALINE

No.	Case	Age	Analyses of Stools				Retention of Fat			
			Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
				Soap	Free Fatty Acids	Neutral Fat				
276	R. L. 2	3 yrs.	10.0	54.5	27.2	18.3	1.82	48.1	46.3	96.3
275	R. L. 1	3 yrs.	15.4	48.0	32.4	19.6	....	48.1	....	....
271	A. W. 2	4 yrs. 1 mo	17.6	40.9	32.1	27.0	1.21	48.1	46.9	97.5
298	E. M. 3	2 yrs. 6 mo.	13.3	37.2	38.3	24.5	2.53	34.1	31.6	92.6
302	F. B. 3	3 yrs. 9 mo.	21.0	33.6	27.7	38.7	2.25	42.5	40.3	94.8
317	K. P.	2 yrs. 8 mo.	9.7	33.0	31.5	35.5	1.23	34.5	33.3	96.4
290	D. R.	3 yrs.	10.5	30.0	17.3	52.7	1.26	44.8	43.5	97.2
350	H. J. 2	1 yr. 4 mo.	14.9	30.0	36.5	33.5	1.35	29.1	27.7	95.4
348	M. J. 3	3 yrs. 4 mo.	19.5	19.4	24.6	56.0	2.06	31.3	29.2	93.3
Average.....			14.7	36.3	29.7	34.0	1.71	39.1	37.4	95.7

The fat percentage of dried weight in the stools (Table 1) is uniformly low, only two values being higher than the average found for normal stools of children on a mixed diet without significant quantities of vegetable fat. The soap is distinctly lower and the neutral fat higher than when vegetable fat was not given, while the fatty acids are but little different. The fat excretion is unusually low and the percentage of the fat intake retained is slightly higher than was found when the fat of the diet was mainly milk fat.

In the group in which the stools were acid (Table 3) the fat percentage of dried weight averages somewhat higher than in the alkaline stools. The distribution of fat, which was determined on the dried

stools, is almost the same as that found in the alkaline stools, but since the stools were acid the soap values would probably have been lower and the fatty acids higher if the stools had been analyzed in the moist state. These stools were all distinctly abnormal in appearance, showing mucus, undigested food or marked evidences of fermentation. The average total fat excretion is much higher and the percent of the fat intake retained is lower than when the stools were alkaline.

TABLE 2.—Fat in Stools and Retention of Fat of Children Receiving from 20 to 35 Per Cent. of the Total Fat Intake as Nut Butter. Stools Acid

No.	Case	Age	Analyses of Stools				Retention of Fat			
			Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
				Soap	Free Fatty Acids	Neutral Fat				
381	H. F. 8	1 yr. 3 mo.	18.9	51.7	23.7	24.6	2.07	43.7	41.6	95.3
285	B. W. 13	2 yrs. 4 mo.	18.6	50.2	26.1	23.7	5.33	51.9	46.6	89.8
278	W. R. 1	4 yrs. 6 mo.	21.7	42.4	32.4	25.2	4.34	35.2	30.9	87.8
286	B. W. 14	2 yrs. 4 mo.	20.9	44.6	24.5	30.9	3.70	51.9	48.2	93.0
349	H. J. 1	1 yr. 4 mo.	12.9	31.8	37.7	30.5	1.10	35.5	34.4	96.9
277	R. L. 3	3 yrs. 1 mo.	8.4	20.8	40.7	38.5	1.18	52.1	50.9	97.5
347	M. J. 2	3 yrs.	34.3	9.3	8.9	81.8	4.44	32.4	28.0	86.3
Average			19.4	35.8	27.7	36.5	3.17	43.2	40.0	92.6

The findings when the vegetable fat formed from 35 to 70 per cent. of the total fat intake are given in Tables 3, 4 and 5. In the instances reported in Table 3 the vegetable fat was nut butter, in those in Tables 4 and 5 it was corn oil. None of the stools of the children receiving this proportion of nut butter were acid. In three instances when the fat was corn oil there were acid stools (Table 5).

TABLE 3.—Fat in Stools and Retention of Fat of Children Receiving from 35 to 70 Per Cent. of the Total Fat Intake as Nut Butter. Stools Alkaline

No.	Case	Age	Analyses of Stools				Retention of Fat			
			Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
				Soap	Free Fatty Acids	Neutral Fat				
384	F. B. 6	3 yrs. 10 mo.	14.4	51.5	20.6	27.9	2.09	40.9	38.8	94.9
264	F. W. 9	3 yrs. 7 mo.	16.3	49.8	37.1	13.1	2.92	43.6	40.7	93.3
361	R. L. 8	4 yrs. 3 mo.	9.7	45.4	23.7	30.9	1.75	45.4	43.7	96.1
274	A. W. 5	4 yrs. 2 mo.	13.6	41.4	31.6	27.0	2.56	52.1	49.5	95.2
379	H. F. 4	1 yr. 1 mo.	20.8	40.2	31.3	28.5	2.56	37.3	34.7	93.1
382	E. M. 5	2 yrs. 6 mo.	13.3	35.9	29.1	35.0	1.84	39.2	37.4	95.3
360	R. L. 6	4 yrs. 2 mo.	10.8	31.0	25.4	43.6	1.86	43.2	41.3	95.7
Average			14.1	42.2	28.4	29.4	2.23	43.1	40.9	94.9

The fat percentage of dried weight (Table 3) is much the same in range and average as (Table 1) when the diet contained a smaller proportion of nut butter. The average for distribution of fat in the stools is almost the same as when the fat of the diet was chiefly milk fat. The average fat excretion is slightly less and the percentage retention is slightly higher with this proportion of nut butter than when the fat in the diet was mainly milk fat.

TABLE 4.—Fat in Stools and Retention of Fat of Children Receiving from 35 to 70 Per Cent. of the Total Fat Intake as Corn Oil. Stools Alkaline

No.	Case	Age	Analyses of Stools				Retention of Fat			
			Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
				Soap	Free Fatty Acids	Neutral Fat				
366	W. R. 3	4 yrs. 7 mo.	26.5	48.6	24.5	26.9	4.66	77.0	72.3	94.0
364	O. W. 4	2 yrs. 7 mo.	19.7	44.1	23.9	32.0	2.10	47.5	45.4	95.6
272	A. W. 3	4 yrs. 1 mo.	20.6	40.4	33.2	26.4	2.20	52.1	49.9	95.8
273	A. W. 4	4 yrs. 1 mo.	20.6	36.2	33.8	30.0	5.07	52.1	47.0	90.3
367	W. R. 4	4 yrs. 7 mo.	24.0	34.8	22.3	42.9	3.87	77.0	73.1	95.0
369	W. R. 6	4 yrs. 9 mo.	12.2	29.1	38.6	32.3	1.50	63.1	61.6	97.6
Average			20.6	38.9	29.4	31.7	3.23	61.4	58.2	94.6

When the vegetable fat was corn oil, forming from 35 to 70 per cent. of the total fat intake, the average fat percentage of dried weight in the alkaline stools (Table 6) is slightly higher than that which was found when the fat of the diet was mainly milk fat. The distribution of fat is similar to that found with milk fat feeding, though the soap is a little lower and the neutral fat correspondingly higher. The fat excretion averages considerably higher than when the diet contained no vegetable fat. The total fat intake is very high, making the actual retention high and the percentage retention about normal.

TABLE 5.—Fat in Stools and Retention of Fat of Children Receiving from 35 to 70 Per Cent. of the Total Fat Intake as Corn Oil. Stools Acid

No.	Case	Age	Analyses of Stools				Retention of Fat			
			Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
				Soap	Free Fatty Acids	Neutral Fat				
363	O. W. 3	2 yrs. 7 mo.	16.0	43.1	40.6	16.3	1.31	47.5	46.2	97.2
365	W. R. 2	4 yrs. 7 mo.	14.9	37.7	35.7	26.6	2.57	64.5	61.9	96.0
359	R. L. 4	3 yrs. 1 mo.	27.4	36.4	49.0	14.6	3.94	63.6	59.7	93.8
Average			19.4	39.1	41.7	19.2	2.61	58.5	55.9	95.6

In the acid stools (Table 5) the fat percentage of dried weight averages about the same as in the alkaline stools when the diet contained the same proportions of corn oil. A difference is seen in the distribution of fat, the fatty acids being considerably increased at the expense of the neutral fat. There is a smaller average fat loss and a slightly higher percentage retention.

In Tables 6 and 7 are shown the findings when the vegetable fat was 70 to 95 per cent. of the total fat intake. Table 6 gives the values when the fat was nut butter; Table 7 when it was corn oil. The stools were all alkaline, except in one instance with corn oil feeding. The values in this case are placed in the same table with the values for the alkaline stools but are not included in the average.

TABLE 6.—FAT IN STOOLS AND RETENTION OF FAT OF CHILDREN RECEIVING FROM 70 TO 95 PER CENT. OF THE TOTAL FAT INTAKE AS NUT BUTTER. STOOLS ALKALINE

No.	Case	Age	Analyses of Stools				Retention of Fat			
			Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
				Soap	Free Fatty Acids	Neutral Fat				
362	R. L. 10	4 yrs. 3 mo.	8.9	47.0	19.9	33.1	1.68	44.6	42.9	96.2
383	E. M. 7	2 yrs. 8 mo.	14.9	43.9	28.0	28.1	2.37	38.1	35.7	93.8
380	H. F. 6	1 yr. 2 mo.	18.4	33.2	37.0	29.8	2.24	35.3	33.1	93.7
385	F. B. 8	3 yrs. 10 mo.	13.3	28.9	21.4	49.7	1.42	41.8	40.4	96.6
Average			13.9	38.2	26.6	35.2	1.93	39.9	38.0	95.2

Table 6 shows that when the diet contained this high proportion of the fat as nut butter the fat percentage of dried weight and the distribution of fat in the stools are not significantly different from that found when the proportion of nut butter was less. The average total fat in the stools is less and the percentage retention slightly higher than when the fat intake consisted mainly of milk fat.

When the high proportion of corn oil was given the fat percentage of dried weight of the stools (Table 7) is very high, the average being 31.7 per cent. The soap is lower than in any other group in which the vegetable fat formed a significant part of the fat intake. The fat loss is the greatest seen with the vegetable fat feeding, but since the intake is high, the actual retention is still very high and the percentage of the intake retained is good, although lower than in the other groups.

Table 8 gives a comparison of the averages for the various groups here presented with the averages found when the diet contained no significant proportion of the fat as vegetable fat, both when the stools were normal and when they were acid.

TABLE 7.—Fat in Stools and Fat Retention of Children Receiving from 70 to 95 Per Cent. of the Total Fat Intake as Corn Oil. Stools Alkaline

No.	Case	Age	Analyses of Stools				Retention of Fat			
			Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
				Soap	Free Fatty Acids	Neutral Fat				
371	F. W. 5	3 yrs. 6 mo.	36.5	37.6	34.9	27.5	5.30	59.4	54.1	91.1
376	B. W. 9	2 yrs. 2 mo.	24.8	37.0	36.3	26.7	....	44.5	....	....
370	F. W. 4	3 yrs. 6 mo.	47.2	34.6	46.9	18.5	4.01	59.4	55.4	93.2
375	B. W. 8	2 yrs. 1 mo.	40.0	34.5	26.6	38.9	7.01	49.8	42.8	86.0
374	B. W. 7	2 yrs. 1 mo.	31.1	32.0	28.5	39.5	6.44	49.7	43.3	87.1
372	F. W. 6	3 yrs. 7 mo.	24.6	21.6	29.7	48.7	3.03	59.4	56.4	94.9
373	F. W. 7	3 yrs. 7 mo.	17.8	14.6	49.7	35.7	3.01	59.4	56.4	94.9
Average.....			31.7	30.3	36.0	33.7	4.80	56.2	51.4	91.5
	Stools Acid									
358	C. H.	6 yrs.	13.2	31.8	32.6	35.6	....	35.2	....	....

TABLE 8.—Comparison of Fat in Stools and Fat Retention of Children Receiving Milk Fat with That of Children Receiving Varying Proportions of Corn Oil and of Nut Butter

Fat in Diet	Reaction of Stools	Analyses of Stools				Retention of Fat			
		Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
			Soap	Free Fatty Acids	Neutral Fat				
Fat mostly as milk or butter	Alkaline	18.0	45.1	27.4	27.5	2.48	40.8	38.3	93.9
	Acid	15.1	32.8	31.3	35.9	2.38	40.1	37.7	94.0
20 to 35% of fat as nut butter	Alkaline	14.7	36.3	29.7	34.0	1.71	39.1	37.4	95.7
	Acid	19.4	35.8	27.7	36.5	3.17	43.2	40.0	92.6
35 to 70% of fat as nut butter	Alkaline	14.1	42.2	28.4	29.4	2.23	43.1	40.9	94.9
	Acid	19.4	39.1	41.7	19.2	2.61	58.5	55.9	95.6
35 to 70% of fat as corn oil	Alkaline	20.6	38.9	29.4	31.7	3.23	61.4	58.2	94.6
70 to 95% of fat as nut butter	Alkaline	13.9	38.2	26.6	35.2	1.93	39.9	38.0	95.2
70 to 95% of fat as corn oil	Alkaline	31.7	30.3	36.0	33.7	4.80	56.2	51.4	91.5

When the vegetable fat in the diet was nut butter in whatever proportion, the fat in the alkaline stools averages about 14 per cent. of the dried weight. In the group of acid stools with nut butter feeding the fat percentage of dried weight is higher than in the alkaline stools, which is the opposite of what was found with general mixed diet. When a moderate proportion of corn oil was included in the diet, the fat percentage of dried weight in the stools is about the same as when there was no vegetable fat in the diet, whether the stools were acid or alkaline. When, however, the amount of corn oil was large and formed the major part of the fat intake, the fat percentage of dried weight is very high for mixed feeding, averaging 31.7.

TABLE 9.—Results of Feeding with Corn Oil

Case 1.—O. W., aged 2½ years. Healthy child, fairly well nourished. Initial weight, 10,120 gm. Final weight, 10,618 gm.

Period	Fat in Diet	Length of Period, Days	Average Daily Gain or Loss in Weight, Gm.	Appetite	Condition	Stools		No.	Analyses of Stools				Retention of Fat			
						Character	Number Daily		Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
										Soap	Free Fatty Acids	Neutral Fat				
1	83% of fat as milk	30	+ 4	Good	Improving from slight disturbance of digestion	Good, occasionally hard	1-2	268	29.8	64.6	16.5	18.9	2.16	38.5	36.3	94.4
2	53% of fat as corn oil, 34% of fat as milk (skimmed)	18	+27	Good	Excellent	Good, occasionally hard	1-2	364	19.7	44.1	23.9	32.0	2.10	47.5	45.4	95.6
.....	.....	17	....	.....	Measles, severe attack											
3	28% of fat as cod liver oil, 60% of fat as milk	7	+ 5	Good	Good	Good, occasionally hard	1	269	21.8	53.0	28.2	18.8	3.20	53.5	50.3	94.0

TABLE 10.—Results of Feeding with Corn Oil and Nut Butter

Case 2.—A. W., aged 4 years. Healthy, well nourished child. Initial weight, 14,830 gm. Final weight, 15,540 gm.

1	83% of fat as milk	19	—36	Good	Good, slightly constipated	Good	0-2	270	13.4	47.1	29.7	23.2	1.43	38.4	37.0	96.3
2	33% of fat as nut butter, 50% of fat as milk	13	+65	Good	Excellent, slightly constipated	Good	0-1	271	17.6	40.9	32.1	27.0	1.21	48.1	46.9	97.5
3	38% of fat as corn oil, 46% of fat as milk	24	+24	Good	Excellent	Good, occasionally hard	1	272	20.6	40.4	33.2	26.4	2.19	52.1	49.9	95.8
								273	20.6	36.2	33.8	30.0	5.07	52.1	47.0	90.3
...	.....	13	....	.....	Measles, mild attack											
4	38% of fat as nut butter, 46% of fat as milk	7	+38	Good	Good, slightly constipated	Constipated	1	274	13.6	41.4	31.6	27.0	2.56	52.1	49.5	95.2

TABLE 11.—Results of Feeding with Corn Oil for Long Period with Egg

Case 3.—F. W., aged 3 years, 3 months. Well nourished child, previously very rachitic. Initial weight, 9,500 gm. Final weight, 11,120 gm.

1	35% of fat as cod liver oil. 53% of fat as milk	31	+23	Fair	Excellent, gaining rapidly in strength	Good	1	263	32.1	59.6	19.7	20.7	3.44	56.7	53.3	94.0
	Corn oil gradually substituted for milk fat and cod liver oil	23	....	.....	Excellent											
2	84% of fat as corn oil (fat-free milk)	46	20+	Fair	Excellent, much more active	Good, occasionally not formed	1-2	370	47.2	34.6	46.9	18.5	4.01	59.4	55.4	93.2
								371	36.5	37.6	34.9	27.5	5.30	59.4	54.1	91.1
								372	24.6	21.6	29.7	48.7	3.03	59.4	56.4	94.9
								373	17.8	14.6	49.7	35.7	3.01	59.4	56.4	94.9
.....	.....	9	....	.....	Measles, mild attack											
3	37% of fat as nut butter, 46% of fat as milk	9	+44	Good	Excellent	Good, sometimes loose	1-2	264	16.3	49.8	37.1	13.1	2.92	43.6	40.7	93.3

TABLE 12.—Results from Replacing Milk Fat by Corn Oil without Egg

Case 4.—B. W., aged 2 years. Healthy, well nourished child. Initial weight, 10,610 gm. Final weight, 11,710 gm.

Period	Fat in Diet	Length of Period, Days	Average Daily Gain or Loss in Weight, Gm.	Appetite	Condition	Stools		No.	Analyses of Stools				Retention of Fat			
						Character	Number Daily		Fat Percentage of Dried Weight	Percentage of Total Fat as Soap	Free Fatty Acids	Neutral Fat	Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
1	81% of fat as milk	25	+11	Good	Excellent	Good	1-3	283	24.0	55.5	19.5	25.0	3.36	42.1	38.7	92.0
	Corn oil gradually substituted for milk fat	7	....	.....	Excellent											
2	95% of fat as corn oil (fat-free milk)	39	+13	Good	Good, urticaria on legs during two weeks. Inflammation of eyelids appeared twice, developing once into stye	Good	1-2	374	31.1	32.0	28.5	39.5	6.44	49.7	43.3	87.1
								375	40.0	34.5	26.6	38.9	7.01	49.8	42.8	86.0
								376	24.8	37.0	36.3	26.7	....	44.5	....	....
3	95% of fat as corn oil (fat-free milk) (1 oz. chalk mixture)	13	+11	Good	Excellent	Mushy, then good	1	377	20.6	33.9	25.2	40.9	4.65	44.5	39.8	89.6
								378	17.5	40.9	28.6	30.5	3.30	44.5	41.2	92.7
.....	.....	15	....	.....	Measles, mild attack											
4	79% of fat as milk	8	+29	Good	Excellent	Constipated	1	284	17.8	46.3	26.9	26.8	3.85	40.5	36.7	90.5
5	{31% of fat as nut butter, {54% of fat as milk	9	+21	Good	Excellent	Constipated, then acid, not good	1	285	18.6	50.2*	26.1*	23.7	5.33	51.9	46.6	89.8
								286	20.9	44.6*	24.5*	30.9	3.70	51.9	48.2	93.0

TABLE 13.—Results of Feeding with Corn Oil When Curd Is Included in Diet

Case 5.—W. R., aged 4½ years. Fairly well nourished child, suffering from chronic intestinal indigestion. Initial Weight, 14,750 gm. Final weight, 15,950 gm.

1	{24% of fat as nut butter, {49% of fat as milk	5	−46	Good	Fair	Fair, acid	0-2	278	21.7	42.4*	32.4*	25.2	4.34	35.2	30.9	87.8
	Corn oil gradually added to diet	13	....	.....	Fair											
2	{54% of fat as corn oil, {31% of fat as milk	22	+11	Good	Good	Loose, fermentative	1-2	365	14.9	37.7*	35.7*	26.6	2.57	64.5	61.9	96.0
3	{45% of fat as corn oil, {43% of fat as milk	10	+53	Good	Good, improving	Generally thin with mucus	1-2	366	26.5	48.6	24.5	26.9	4.66	77.0	72.3	94.0
								367	24.0	34.8	22.3	42.9	3.87	77.0	73.1	95.0
4	{42% of fat as corn oil, {44% of fat as milk (2 oz. chalk mixture)	8	−17	Good	Good, except for slight bronchial cold	Thin	1	368	25.4	54.4	19.3	26.3	4.74	60.1	55.4	92.1
5	{40% of fat as corn oil, {40% of fat as milk	21	+17	Fair	Improving	Mostly thin	1	369	12.2	29.1	38.6	32.3	1.50	63.1	61.6	97.6

* Stools acid.

TABLE 14.—Results of Feeding Corn Oil and Nut Butter

Case 6-A.—R. L., aged 3 years. Neuropathic child, normal as to digestion. Initial weight, 12,050 gm. Final weight, 13,520 gm.

Period	Fat in Diet	Length of Period, Days	Average Daily Gain or Loss in Weight, Gm.	Appetite	Condition	Stools		No.	Analyses of Stools				Retention of Fat			
						Character	Number Daily		Fat Percentage of Dried Weight	Percentage of Total Fat as Soap	Free Fatty Acids	Neutral Fat	Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
1	31% of fat as nut butter, 51% of fat as milk	28	+30	Good	Nervous condition improving	Good	0-3	276	10.0	54.5	27.2	18.3	1.82	48.1	46.3	96.3
								277	8.4	20.8*	40.7*	38.5	1.18	52.1	50.9	97.5
2	47% of fat as corn oil. 38% of fat as milk	12	+13	Good	Much improved.....	Good, sometimes thin	1-3	359	27.4	36.4*	49.0*	14.6	3.94	63.6	59.7	93.8

Case 6-B.—R. L., aged 4 years. Neuropathic child, normal as to digestion. Initial weight, 14,820 gm. Final weight, 14,830 gm.

1	40% of fat as butter, 40% of fat as milk	27	+ 4	Poor	Fair.....	Good, sometimes loose	1	353	20.7	54.1	22.3	23.6	3.57	40.0	36.4	91.1
2	37% of fat as nut butter, 44% of fat as milk	12	— 2	Fair	Improving.....	Good	1-2	360	10.8	31.0	25.4	43.6	1.86	43.2	41.3	95.7
3	60% of fat as butter, 21% of fat as milk (skimmed)	9	+14	Poor	Improving.....	Good, sometimes soft	1-2	354	16.7	41.0	26.4	32.6	3.29	43.2	39.9	92.4
4	57% of fat as nut butter, 25% of fat as milk (skimmed)	7	— 8	Fair	Improving.....	Good, partly not formed	1-2	961	9.7	45.4	23.7	30.9	1.75	45.4	43.7	96.1
5	81% of fat as butter (Fat-free milk)	9	+67	Good	Good.....	Good	1-2	355	15.5	32.3	23.5	44.2	3.34	44.6	41.3	92.5
6	81% of fat as nut butter (Fat-free milk)	7	+ 8	Good	Good.....	Good	1	362	8.9	47.0	19.9	33.1	1.68	44.6	42.9	96.2

TABLE 15.—No Relation Shown to Kind or Amount of Fat

Case 7.—E. M., aged 2½ years. Well nourished child, healthy except for general impetigo contagiosa. Initial weight, 12,788 gm. Final weight, 14,505 gm.

1	84% of fat as milk	13	+37	Fair	Improving.....	Good	1-2	296	16.1	28.9	37.5	33.6	2.29	38.8	36.5	94.1
2	24% of fat as butter, 64% of fat as milk	16	+13	Good	Impetigo cured. Abscess on thigh	Good, partly not formed	2-3 then 1	297	16.0	35.3	31.7	33.0	1.95	32.6	30.6	94.0
3	23% of fat as nut butter, 66% of fat as milk	28	+27	Good	Good, except for short attack of influenza	Good	2-4	298	13.3	37.2	38.3	24.5	2.53	34.1	31.6	92.6
4	50% of fat as butter, 28% of fat as milk (skimmed)	48	0	Poor	Tonsillitis and influenza during part of period	Good	0-3	299	17.4	35.6	33.1	31.3	2.53	40.1	37.6	93.7
5	51% of fat as nut butter,	9	+37	Fair	Good.....	Good	1-2	382	13.3	35.9	29.1	35.0	1.84	39.2	37.4	95.3
.....	.....	17	....	.....	Tonsils and adenoids removed											
6	80% of fat as butter (Fat-free milk)	7	+35	Fair	Fair. Abscess on lip at beginning of period	Good	1	254	16.7	52.6	22.6	24.8	2.94	37.7	34.8	92.2
7	79% of fat as nut butter (Fat-free milk)	6	+17	Good	Good.....	Mostly good	2-3	383	14.9	43.9	28.0	28.1	2.37	38.1	35.7	93.8

* Stools acid.

* Stools acid.

TABLE 16.—Results of Feeding Nut Butter and Milk Butter

Case 8.—F. B., aged 3½ years. Well nourished, healthy child. Initial weight, 15,280 gm. Final weight, 15,265 gm.

Period	Fat in Diet	Length of Period, Days	Average Daily Gain or Loss in Weight, Gm.	Appetite	Condition	Stools		No.	Analyses of Stools				Retention of Fat			
						Character	Number Daily		Fat Percentage of Dried Weight	Percentage of Total Fat as			Total Fat in Feces, Gm. Daily	Intake of Fat, Gm. Daily	Fat Retained, Gm. Daily	Percentage of Intake Retained
										Soap	Free Fatty Acids	Neutral Fat				
1	81% of fat as milk	9	+11	Good	Excellent.....	Good	1-3	300	24.4	70.2	11.9	17.9	2.16	34.3	32.1	93.6
2	{28% of fat as butter, {55% of fat as milk	16	+27	Good	Excellent.....	Good	1-2	301	21.6	49.5*	20.4*	30.1	3.53	43.4	39.9	91.9
3	{28% of fat as nut butter, {55% of fat as milk	17	+ 3	Poor	Fair, slight attack of influenza, furuncle on leg	Good	1-3	302	21.0	33.6	27.7	38.7	2.25	42.5	40.3	94.8
		51	....	.....	Influenza and tonsillitis, tonsils and adenoids removed											
4	{54% of fat as butter, {26% of fat as milk (skimmed)	7	+86	Good	Good, rapidly improving	Good, partly not formed	2	351	20.4	53.5	17.2	29.3	2.74	44.4	41.7	93.8
5	{59% of fat as nut butter, {24% of fat as milk (skimmed)	10	+17	Fair	Otitis media developing	Good, partly not formed	1-2	384	14.4	51.5	20.6	27.9	2.09	40.9	38.8	94.9
6	83% of fat as butter (Fat-free milk)	8	+18	Fair	Fair, otitis media not acute	Good, partly not formed	1-2	352	20.7	49.9	21.2	28.9	2.85	41.1	38.3	93.1
7	81% of fat as nut butter (Fat-free milk)	8	+31	Good	Fair, otitis media becoming acute, inflammation of eyelids, slight skin eruption on upper lip and nostrils	Good	1	385	13.3	28.9	21.4	49.7	1.42	41.8	40.4	96.6

TABLE 17.—Results Not Affected by Kind or Amount of Fat

Case 9.—H. F., aged 1 year. Healthy child, somewhat under weight. Initial weight, 7,260 gm. Final weight, 9,380 gm.

1	86% of fat as milk	19	—13	Good	Fair.....	Constipated	1-2	305	21.5	58.2	22.7	24.1	1.68	34.9	33.2	95.2
	Butter gradually added to diet	13	....	.....	Good											
2	{49% of fat as butter, {36% of fat as milk (skimmed)	11	+30	Good	Good, occasional vomiting	Good, less constipated	1-3	307	18.9	51.2	29.0	19.8	2.55	36.6	34.1	93.0
3	{48% of fat as nut butter, {38% of fat as milk (skimmed)	12	+34	Good	Excellent, slight skin eruption on chin	Good, sometimes hard	1-2	379	20.8	40.2	31.3	28.5	2.56	37.3	34.7	93.1
4	85% of fat as butter (Fat-free milk)	16	+ 8	Good	Fair, slight attack of influenza	Good, sometimes hard	1-2	308	16.3	61.3	18.8	19.9	1.96	35.3	33.3	94.4
5	85% of fat as nut butter (Fat-free milk)	14	+27	Fair	Excellent, eruption on chin	Good	0-2	380	18.4	33.2	37.0	29.8	2.24	35.3	33.1	93.7
6	{30% of fat as butter, {51% of fat as milk	20	+22	Fair	Good, slightly constipated	Good, partly hard	0-2	309	21.0	27.1	29.3	43.6	2.71	39.8	37.1	93.2
7	{28% of fat as nut butter, {55% of fat as milk	17	+ 9	Fair	Excellent.....	Constipated, then good	1-2	381	18.9	51.7*	23.7*	24.6	2.07	43.7	41.6	95.3

* Stools acid.

The distribution of fat in the stools when the diet contained a considerable proportion of vegetable fat shows no striking variation from the findings when the diet did not contain vegetable fat in significant amounts. The soap percentage is usually somewhat lower and the neutral fat somewhat higher. When most of the fat intake was corn oil the soap percentage shows a lower average than that in any of the other groups. The only unusual value for fatty acids is seen in the group of acid stools with corn oil feeding. Here the fatty acids are much increased at the expense of the neutral fat.

The fat excretion in the alkaline stools when the vegetable fat was nut butter is somewhat less than when the fat intake was mainly milk fat. When the vegetable fat was corn oil the fat loss is greater, being much increased when a large amount of corn oil was given. However, the total fat intake of the children receiving corn oil was very high. In all the groups with vegetable fat feeding the actual retention of fat is never appreciably less than when the diet did not contain vegetable fat and is much greater when the total fat intake was high. In only two groups is the average percentage retention lower than the normal value found with mixed diet without vegetable fat and in these it is over 90 per cent.

PART II.

In order to study the effect on the composition of the stools, the fat retention and the general condition when vegetable fat is substituted for milk fat in varying proportions in a mixed diet, nine children were observed for long periods. In reporting the results of this study each child is considered separately. A table is given which shows the fat percentage and the distribution of fat in the stools and the retention of fat with varying conditions of fat intake, some clinical observations also being included.

These children all received a general mixed diet, usually including an egg daily or alternately egg and meat. The changes in the kind of fat intake are shown in the tables.

CASE 1.—O. W. The findings in this case showed that when a large proportion of the intake was corn oil the fat retention was excellent and the fat percentage and distribution of the fat in the stools not significantly different from the average found for mixed diet without vegetable fat.

CASE 2.—A. W. This child showed excellent digestion of vegetable fat, both nut butter and corn oil, the percentage of the intake retained being usually considerably higher than the average found for general mixed diet. In one instance with corn oil feeding there was an unusually large excretion and consequently lower percentage retention, but even in this instance the actual retention was high. The fat percentage and distribution of fat varied little in different observations, whatever the kind of fat.

CASE 3.—F. W. This child received a diet including 50 gm. of corn oil for a long period. During this time the only sources of fat soluble vitamin

were the daily egg and the traces of fat left in the milk, unless, as has been supposed by some, corn oil contains more of this constituent than do the other vegetable oils. The gain in weight and strength continued and the utilization of this large amount of corn oil was good. In observations 370 and 371 the total fat and the fat percentage of dried weight in the stools were unusually large for mixed diet, but with a high intake, the actual retention was much greater than the average for mixed diet without vegetable fat.

CASE 4.—B. W. In this case also the milk fat in the diet was replaced by a large amount of corn oil. Then egg was omitted from the diet, so that for a considerable period there was no definite source of fat-soluble vitamin. The child continued in excellent health and condition, although a week after the egg was omitted from the diet he ceased to gain in weight. During the early part of the long corn oil period he had styes and an attack of urticaria but he was free from these during the later weeks. The stools of this child were habitually larger, whatever the diet, than is usual for his age. The excretion of fat was always high and the percentage of the intake retained never as high as the normal average for mixed diet. Except in the first observations with corn oil feeding, the percentage of the fat intake retained was as good with either corn oil or nut butter as with milk fat. Since with corn oil feeding the soap percentage of total fat in the stools was lower than with milk fat feeding an attempt was made to increase the proportion of soap in the stool by the administration of one ounce of chalk mixture daily. This had very little effect. This child, whose retention of all fats was below normal, showed nearly as good utilization of corn oil and nut butter as of milk fat.

CASE 5.—W. R. This child before coming under our observation was receiving a diet containing little fat and a large proportion of carbohydrates. He had a chronic intestinal indigestion with acid fermentative stools. When the first observation in this series was made he was taking the diet mentioned. The percentage of the fat intake retained was somewhat below the normal average for mixed diet. The proportion of carbohydrates in the diet was reduced and the fat greatly increased, partly by corn oil. Later both the fat and the protein were increased by adding curd from whole or partially skimmed milk. Although the stools continued to be thin and frequently showed evidences of fermentation, the condition of the child improved and there was a very good gain in weight. The percentage of the fat intake retained, except in one period when he had a severe cold, was higher than the normal average. Although the fat loss in the stools was usually greater than the average for mixed diet, the actual retention was extremely high, because of the unusually large intake of fat. The fat percentage and distribution of fat in the stools showed considerable variation, apparently not related to the kind of fat in the diet. In observation 367 the soap percentage of total fat was low and the neutral fat high. An attempt was made to increase the proportion of soap by administering two ounces of chalk mixture daily and the next observation showed the highest soap value in the series. Although this child had stools which were looser than normal and frequently fermentative he retained a large proportion of a very high intake, nearly half of which was corn oil.

CASE 6-A.—R. L. This child showed excellent retention of fat both when nut butter and when corn oil formed part of the fat intake. The percentage of the intake retained was better with nut butter, but with a larger intake the actual retention was better with corn oil. The fat percentage of dried weight in the stools and the total fat excretion were higher with corn oil feeding than when nut butter was taken.

In the succeeding cases the same general plan was followed. A preliminary observation was made with a diet containing a quart of milk but no butter. The amount of milk was then reduced to about 20 ounces and a moderate amount, from 8 to 16 gm., of milk butter was added to the diet. The milk butter was then replaced by an equal amount of nut-butter. Partially skimmed milk, with about 1.8 per cent. of fat, was then given and a larger amount, from

18 to 26 gm., of butter given, first milk butter and then nut butter. Finally fat-free milk was given with a still larger amount, from 30 to 36 gm., first of milk butter and then of nut butter.

CASE 6-B.—R. L. In this case two points are to be noted in regard to fat percentage of dried weight in the stools. First, as the proportion of fat in the form of butter increased, the fat percentage of dried weight in the stools decreased. Second, when nut butter replaced the milk butter, the fat percentage of dried weight was much lower than with the same proportion of milk butter. The fat excretion was about twice as great when milk butter was given as when nut butter was given and the percentage of the fat intake retained considerably higher with the nut butter than with the corresponding amount of milk butter. This child showed excellent digestion of nut butter but there was little or no gain in weight during the periods when nut butter was taken.

CASE 7.—E. M. With this child the fat excretion was never large and showed no constant relation to the kind or amount of fat. The percentage of the fat intake retained was always fairly high.

CASE 8.—F. B. In this case, the fat excretion when nut butter was included in the diet was less than the normal average for mixed diet and the percentage of the fat intake retained higher than normal. With milk butter the fat loss was somewhat greater and the percentage of the intake retained slightly lower.

CASE 9.—H. F. With this child the fat excretion was never large and the percentage of the fat intake retained always good, regardless of the amount and kind of fat in the diet. This child digested milk butter and nut butter equally well.

In general it can be said that in no single instance was there any evidence that the vegetable fats given caused any disturbance of digestion. No child showed any impairment of appetite. The children took these fats well, in some cases eagerly, and in no case was there vomiting.

The stools of the children taking the large amounts of vegetable fats were, as a rule, rather softer than when mainly milk fat was taken, this being more marked with the corn oil feeding than with nut butter. The frequency of the stools was not increased. In no instance were loose stools seen which could be definitely attributed to the vegetable fat. In the case of W. R., who was suffering from chronic intestinal indigestion, the stools were more loose after the first period, but the fat intake was so greatly increased that the change in the stools cannot be necessarily attributed to the corn oil.

It is to be noted that one child, F. W., took 50 gm. of corn oil daily for forty-six days; another, B. W., took an average of 45 gm. daily for fifty-two days, and a third, W. R., took 35 gm. daily for thirty-two days, all without any disturbing effects.

The increase in weight was, on the whole, quite as uniform and as good with nut butter and corn oil as with milk fat, the only exception being in the case of R. L. (Case 6-B). With this child the decided gain noted in the fifth period was perhaps due to an increase in the amount of food given, coincident with an improvement in the appetite. The marked gain noted following an attack of measles in three

instances is to be explained by the fact that during the acute illness the food had been much reduced and the subsequent increase in weight was the gain of convalescence.

SUMMARY

1. The stools of children receiving a considerable proportion of vegetable fat did not differ essentially in appearance from those of children receiving mainly milk fat, although they were usually somewhat softer.

2. The fat percentage of dried weight of the stools averaged somewhat lower when nut butter was taken, and somewhat higher when corn oil was taken, than when the fat in the diet was mainly milk fat; and when large quantities of corn oil were included in the diet the average was much higher.

3. The soap percentage of total fat in the stools was usually a little lower and the neutral fat a little higher with vegetable fat than when the fat of the diet was mainly milk fat.

4. When nut butter was taken the fat excretion in the alkaline stools was lower and in the acid stools it was higher, than when the diet did not contain vegetable fat. When corn oil was taken in considerable amounts the fat excretion in the stools was higher than when the fat of the diet was mainly milk fat. However, the total fat intake when corn oil was included in the diet was very large and the actual retention of fat always much higher than the normal average for mixed diet. When vegetable fat formed a considerable part of the total fat intake, the percentage of the fat intake retained was usually higher than the normal average. In a few instances when the stools were acid and in a few when large amounts of corn oil were taken, the percentage retained was low.

5. The individual children observed for considerable periods with changes in the kind and amount of fat intake showed quite as good digestion of vegetable fat as of corresponding amounts of milk fat and no unfavorable effect on general health and nutrition was observed. No children were kept long enough on a diet presumably deficient in fat-soluble A to warrant any conclusions as to the effect of such a diet upon growth and health. In the case of one child who for five weeks was on a diet in which there was no definite source of fat-soluble vitamin, .95 per cent. of the fat of the diet being corn oil, he ceased to gain in weight, but showed no loss and the general health continued excellent. The fact may not be without significance that of six children, 80 to 95 per cent. of whose fat intake was vegetable fat, two developed styes and two others eczema upon the face, which disappeared when the diet was changed to include milk fat.

CONCLUSION

The observations published in this paper indicate that corn oil and nut butter, the vegetable fats studied, are valuable foods for children, are exceedingly well borne and are apparently digested and absorbed with ease. We feel, therefore, warranted in the belief that these articles may safely be introduced into the regular diet of children, and that to a considerable degree they may be substituted for the more expensive milk fat, given either as milk or as butter, but they should never entirely replace milk fat. How much milk fat is needed to furnish the amount of the fat-soluble vitamin required for normal growth and nutrition we have not yet the data to determine.

CEREBROSPINAL INVOLVEMENT IN HEREDITARY SYPHILIS *

PHILIP C. JEANS, M.D.
ST. LOUIS

In this paper it is intended to show that contrary to most current opinion, involvement of the central nervous system in hereditary syphilis is relatively common, and that by statistical study there is no difference in the incidence of central nervous involvement in hereditary and in acquired syphilis. It is further emphasized that the treatment for neurosyphilis is the same and should be carried out just as intensively whether the infection is acquired or hereditary. As a matter of fact, the differences between hereditary and acquired syphilis are accounted for by the fact that in one case it occurs in a rapidly developing organism and in the other case in a mature organism.

There have appeared in the last few years a number of studies of the incidence of cerebrospinal involvement in acquired syphilis based on the examination of the cerebrospinal fluid. Most of these studies have been made on patients having syphilis it its early stages. There is practically nothing in the literature in regard to similar observations on patients with hereditary syphilis. There is a priori no good reason why hereditary and acquired syphilis should differ in this respect. In either case there is an excellent opportunity for the central nervous system to become involved during the general dissemination of the spirochetes over the body.

Neurologists commonly claim that not more than from 5 to 10 per cent. of individuals with acquired syphilis develop neurosyphilis. Estimates based entirely on clinical observation necessarily represent a minimum because they do not include those cases with vaguely defined clinical signs which we now know to be so frequently associated with abnormalities of the cerebrospinal fluid. Probably the largest and best studied series of cases is that of Mattauschek and Pilz[1] in which 4,134 army officers were followed for from ten to thirty years. In that period 10.25 per cent. had developed paresis, tabes or cerebrospinal syphilis.

---

* From the Department of Pediatrics, Washington University School of Medicine.

* Read before the American Pediatric Society, June, 1919.

1. Mattauschek and Pilz: Quoted by Fordyce, J. A. M. A. **71**:1023 (Sept. 28) 1918.

Since the era of laboratory diagnosis the various studies of the cerebrospinal fluid in acquired syphilis have generally shown a higher incidence of central nervous involvement than was found by clinical observation. There are certain difficulties with such diagnosis that need to be understood before such studies can be interpreted. A positive Wassermann reaction in the cerebrospinal fluid is not observed until the serum is positive, and even then it is usually delayed. Most of the recent laboratory studies include such early cases or are almost exclusively made up of them, thus making that most valuable laboratory test almost useless. At this early stage the diagnosis must be based largely on the increase in cells, globulin and albumen, and on the colloidal gold test. In many instances these changes are so conspicuous as to leave no doubt of the diagnosis, but there is always a large group of borderline cases in which the changes are inconclusive. Studies among individuals who have had syphilis for several years show a more clean cut differentiation between the positive and negative cases and give a better idea as to the proportion that will develop neurosyphilis.

Slight variations in the cerebrospinal fluid from the normal have been reported by some observers as occurring in the great majority of individuals with early acquired syphilis. These changes have been variously interpreted. Some have regarded them as being a part of a constitutional reaction to the general invasion of the spirochete and therefore in a large measure transitory. Others think that the central nervous system is invaded in the majority of instances and that in many individuals the infection later undergoes spontaneous resolution. Whatever the interpretation, it would seem that only the conspicuous changes in the cerebrospinal fluid have any importance as far as the welfare of the patient is concerned. When such changes are present one can scarcely avoid the conclusion that the central nervous system has been seriously invaded.

A review of the literature shows that in early acquired syphilis cerebrospinal fluid study reveals conspicuous changes in from 25 to 40 per cent. of the cases. The variations in the duration of the infection in these studies and the variation in the standards as to what constitutes conspicuous changes render it difficult to compile from these statistics an average figure on the resulting large number of examinations. However, I think it would be fair to state that practically one third of all individuals with acquired syphilis in the early stages show changes in the cerebrospinal fluid which strongly indicate serious invasion of the central nervous system.

Later in the disease the Wassermann reaction on the cerebrospinal fluid plays a larger rôle in the diagnosis. In most instances there is

either a complete fixation or a complete absence of fixation, thus making a clean cut differentiation. There are but few studies of the cerebrospinal fluid at this stage. Of sixty-four patients observed two years or more after infection, Engman[2] found 34.4 per cent. to have a positive Wassermann reaction in the cerebrospinal fluid and 76 per cent. of these showed clinical symptoms referable to the central nervous system.

Since about one third of all individuals with acquired syphilis show laboratory evidence of involvement of the central nervous system early in the disease and about the same proportion show more definite evidence of such involvement late in the disease, many syphilographers believe, and with justification, that those who show early evidence of central nervous system involvement, and perhaps only those are potential candidates for late neurosyphilitic manifestations.

With[3] and Ravaut[4] each report eight patients with hereditary syphilis examined in the first year of life with the finding of conspicuous cerebrospinal fluid changes in none. These infants are probably more fortunate than the next similar number would have been. However, in twenty-six patients with late hereditary syphilis With found definite pathology in eight, or 31 per cent. No other such statistical studies in hereditary syphilis have come to my attention.

For some time a spinal puncture has been made routinely on all children with syphilis coming to the Washington University Dispensary. Since this measure has been a part of the routine examination, seventy-eight infants under 2 years of age and 136 children over 2 years of age have been studied in regard to nervous system involvement. These patients have had much detailed study that is not evident in this presentation. They have been studied neurologically by Dr. Sidney I. Schwab and fundus examination of the eye has been routinely made. Dr. John Green[5] has already reported the eye findings in more than 100 of these patients. It is intended to report these patients from a neurologic point of view at another time. The chief purpose of these statistics as here presented is to point out the frequency with which the central nervous system is seriously involved in hereditary syphilis.

It is usually conceded that if the cerebrospinal fluid gives a positive Wassermann reaction there is involvement of the central nervous system. The finding of increased cells, globulin, albumen and a positive colloidal gold reaction, though important, has not the same significance as a positive Wassermann. For this reason the Wassermann reaction is especially stressed in this study. The Wassermann reactions

2. Engman: J. A. M. A. **61**:735 (Sept. 6) 1913.
3. With: Brain **40**:403 1917.
4. Ravaut: Quoted by With,[3] Brain **40**:403, 1917.
5. Green: Trans. Am. Ophthal. Soc., June, 1919.

have been made by an expert technician using for each test 0.8 c.c. of spinal fluid and three antigens, simple alcoholic, cholesterinized and Noguchi's acetone insoluble lipoid antigen. By 4 plus is meant complete fixation with all antigens and by one plus is meant positive with the cholerterinized antigen only. The 2 plus and 3 plus are arbitrary gradations between these two extremes.

Of the seventy-eight infants the serum of all but one gave a strongly positive Wassermann reaction. The exception was an infant, 1 month old, who had a rash, rhinitis and infiltration of the palms and soles. The cerebrospinal fluid of thirty-one of these infants gave a positive Wassermann reaction. Of these, eleven reactions were 4 plus, one was 3 plus, ten were 2 plus and nine were 1 plus. Of the nine 1 plus reactions, eight were in infants under 3 months of age, i. e., at a time which might easily be too early for the Wassermann reaction to be strongly manifest even though the nervous system might be definitely involved. In six of these nine instances of 1 plus reaction the cerebrospinal fluid showed other changes corroborating the positive Wassermann reaction. Three were not otherwise examined, but in one a later specimen of cerebrospinal fluid gave a 4 plus Wassermann reaction.

Eleven of the thirty-one infants whose cerebrospinal fluids gave positive Wassermann reactions had clinical signs of neurosyphilis. The signs manifested were chiefly those of meningeal involvement with evidence of deeper involvement in addition in three. Three patients with convulsions were seen at the age of 3, 3 and 10 months, respectively, soon after the onset of the convulsions. One patient with neck rigidity and other signs of meningitis, but without convulsions, was seen soon after onset at the age of 7 months. Four patients with moderate grades of hydrocephalus were seen at the ages of 5½, 6, 9 and 11 months, respectively. Two patients with optic neuritis were seen at the ages of 1½ and 3 months, respectively. One patient with hemiplegia and hydrocephalus was seen at the age of 18 months.

Of the 136 syphilitic children from 2 to 14 years of age, seventy have been classed as having a clinically active infection, and sixty-six as having a latent infection. All but one gave a positive serum Wassermann. The exception was a child who had an acute dementing process and whose cerebrospinal fluid gave complete fixation. The children classed as having a latent infection were for the most part brothers and sisters of children having clinically active syphilis. Some of the children having latent syphilis had scars or similar signs of former lesions, but most of them had no signs or symptoms whatever except a positive Wassermann reaction.

Of the seventy actively syphilitic patients, twenty-two, or 31 per cent., had a positive cerebrospinal fluid Wassermann. Of the sixty-six

latent infections, thirteen of the children, or 20 per cent., had positive cerebrospinal fluid Wassermann reaction. Of the total 136 older children, therefore, thirty-five, or 25.8 per cent. had positive cerebrospinal fluid Wassermann reactions. Of the thirty-five patients having positive cerebrospinal fluids, twenty-five had symptoms or signs referable to the central nervous system. In addition to the twenty-two patients with active syphilis, these twenty-five include three patients with fixed pupils who had no other signs of activity than a positive Wassermann reaction and who were, therefore, classed as latent. Of these twenty-five children having clinical signs of neurosyphilis, twelve had gross mental defect associated. The more superficial or meningeal manifestations occurred as follows: Two patients had acute meningitis with convulsions at 3 and 4½ years of age, respectively; one patient had meningitis without convulsions at 4 years of age; five patients had chronic convulsions which were classed as epilepsy (three of these had associated nervous manifestations); two patients had arrested hydrocephalus (one had associated nervous lesions). The submeningeal manifestations occurred as follows: six patients had hemiplegia at 2, 3, 3½, 4, 5 and 8 years of age, respectively; one patient had spastic quadriplegia at 3 years of age, and one had spastic paraplegia at 3 years of age; optic atrophy was seen five times, twice with associated lesions; fixed pupils without optic atrophy were seen four times; one patient had an acute dementing process with the onset at the age of 6 years; one was seen when 11 years of age with juvenile paresis and two other patients had sensory changes with the cerebrospinal fluid reactions of paresis. Two of these latter three had optic atrophy.

It is seen, then, that the nervous system was involved in 40 per cent. of syphilitic infants, 31 per cent. of older children having active infection and 20 per cent. of older children having latent infection. Of the entire group of 214 infants and children the nervous system was involved in seventy, or 32.7 per cent. Of those having positive cerebrospinal fluids slightly more than one third of the infants and slightly more than two thirds of the older children had clinical manifestations of neurosyphilis at the time of observation.

Whether syphilis is hereditary or acquired, the earlier it is treated the better the prospect for complete cure. This is just as true for neurosyphilis as for infection elsewhere in the body. In the great majority of instances intravenous and intramuscular medication will result in the disappearance of the abnormal findings both in the cerebrospinal fluid and in the blood when such treatment is given early in the disease. Later in the disease these measures are often ineffectual, both clinically and serologically. Even the more drastic procedure of intraspinal therapy, though it may bring about a serologic cure, often leaves the clinical condition unimproved.

We have apparently succeeded in curing, both clinically and serologically, a large proportion of those children with early neurosyphilis, who continued under observation. Every one realizes how discouraging is the prospect of cure or even of benefit in children showing evidence of parenchymatous and in many instances of late meningovascular involvement of the central nervous system. In one sense we have succeeded in curing some of these children. We have treated them until the blood and cerebrospinal fluid are repeatedly negative, but we have not brought back the sight of a child with optic atrophy, nor in most instances have we restored function to a child with paralysis. The time to treat neurosyphilis is before it is clinically manifest. This can be done in at least two thirds of the instances of such involvement in infancy and in a considerable proportion at any age if we but take the trouble to examine the cerebrospinal fluid in any patient who has syphilis, whether there are any clinical indications for it or not. Justice to our syphilitic patients demands that the condition of the central nervous system, as evidenced by the cerebrospinal fluid, be known before they are discharged as cured.

It is a common idea that hereditary neurosyphilis is a more hopeless state than if the infection were acquired and that it cannot be treated as intensively with safety. Such has not been our experience. Because of the fear of a Herxheimer reaction, we have been careful to have the patient well treated with mercury for a short period before starting arsphenamin, but after such a period it has been our custom to administer arsphenamin in full doses and at short intervals. In fact, the treatment of neurosyphilis has been more intensive than if such a lesion were not present, because the consequences of insufficient treatment are more serious. It has further been our custom to resort to intraspinous medication in those instances which did not respond with reasonable promptness to the other measures. We have nothing new to offer in the way of treatment except that we have demonstrated that children with neurosyphilis can be treated with safety by the same intensive methods that are used in other clinics for adults with acquired syphilis.

SUMMARY

According to a survey of the literature there is found involvement of the central nervous system, as shown by the cerebrospinal fluid, in about one third of all individuals with acquired syphilis.

Among the 214 patients of this study having hereditary syphilis, a similar incidence of involvement of the central nervous system has been found.

The treatment of hereditary neurosyphilis need differ in no way from that of acquired neurosyphilis, since it can be carried out safely with the same intensity in both instances.

THE INCIDENCE AND SIGNIFICANCE OF THE RHEUMATIC NODULES IN CHILDREN *

JOSEPH BRENNEMANN, M.D.
CHICAGO

The rheumatic nodules that receive so much attention from British writers on rheumatism in children are almost universally considered a rare condition with us. In all our textbooks there is a striking uniformity of expression that these nodules are common in England, but are rarely seen in this country.

Thus Holt and Howland[1] say: "They are certainly not common in this country, and although we have made it a rule to examine rheumatic patients for them, we have seen them but seldom, and they have been prominent in only eight or ten cases. This has also been the experience of most observers in this country. From published reports, however, they appear to be much more frequent in England." In the 1911 edition of Holt[2] the prominent cases had been placed at "two or three."

Koplik[3] says: "The so-called subcutaneous rheumatic nodules are seen in children less frequently in this country than in England."

Rachford[4] says: "In England these nodules are common. . . . In America, however, they are comparatively rare."

Dunn[5] says: "When present, these subcutaneous nodules are diagnostic of rheumatic fever. Judging by published reports, I should say that they are seen much more commonly in England than in this country, and probably also more often in some parts of this country than in others. They are rarely seen around Boston."

In a recent extensive paper, Murray H. Bass[6] discusses at length erythema nodosum, erythema multiforme, erythema papulatum and purpura, with only a brief mention of rheumatic nodules. Of these

---

* Read before the American Pediatric Society, June, 1919.

1. Holt, L. Emmett, and Howland, John: Diseases of Infancy and Childhood, Ed. 7, New York, D. Appleton & Co., 1916, p. 1152.

2. Holt, L. Emmett: Diseases of Infancy and Childhood, Ed. 6, New York, D. Appleton & Co., 1911, p. 1089.

3. Koplik, Henry: Diseases of Infancy and Childhood, Ed. 3, Philadelphia, Lea & Febiger, 1910, p. 466.

4. Rachford, B. K.: Diseases of Children, New York, D. Appleton & Co., 1912, p. 403.

5. Dunn, Charles Hunter: Pediatrics, Ed. 2, 1917, **2**:329.

6. Bass, Murray H.: The Cutaneous Manifestations of Acute Rheumatic Fever in Childhood, Med. Clin. N. A. **2**:201 (July) 1918.

he says: "Strange to say this particular cutaneous manifestation of rheumatism is but rarely encountered in America, whereas it is one of the common findings in England."

About five years ago, in a paper on rheumatism in children, I expressed this same view as follows: "The rheumatic nodule is apparently a very rare condition in this country. In England, on the other hand, they seem very much more common. Thus Still finds them in nearly one half of all the cases of articular rheumatism in children under 12 years of age in the great Ormond Street Hospital wards; in nearly 30 per cent. of all the cases of chorea, cardiac and articular rheumatism in the wards, and in 10 per cent. of the naturally less serious cases in the outpatient department. We have been told by Still (personal statement) and others that we do not look hard enough, and that we expect something large while they may be very small — no larger than a pinhead. With this in mind, and looking for them constantly, I have seen only four well marked cases in this country." I had seen a fifth case with Pfaundler in Munich, and the enthusiasm with which he attacked the case amply confirmed his statement that these cases were "very rare in Germany, but that they seemed quite common in England."

Before the above paper was published some months later, I added a footnote as follows: "Since writing the above I have seen these nodules so regularly in severe cases of rheumatism with heart involvement that I am inclined to accept Still's figures for our own material from the same social stratum. Thus at the County Hospital at one time all of the children (three in number) with cardiac rheumatism had well marked nodules."

In the last five years since we have been especially interested in these nodules and have really looked for them we have become increasingly convinced that they occur in our cases quite as frequently as they do in an equal number of similar cases of rheumatism in England. There are doubtless more rheumatic nodules to be seen in London than in New York, or Chicago, or Boston, both because rheumatism is more prevalent in England and especially in "the London district" (Voelcker[8]), but even more so because "it is frequently of a severe type," and it is in the severe type of rheumatic infection that the nodules attain their maximum incidence. The views that I have just expressed are fully shared by my colleague, Dr. R. A. Krost, with whom I have worked for many years in the outpatient department and

7. Brennemann, Joseph: Rheumatism in Children, Illinois M. J. **26**:20 (July) 1914.

8. Voelcker, A. F.: Rheumatism and Chorea, Garrod, Batten and Thursfield, Diseases of Children, New York, Longmans, Greene & Co., 1913, p. 95.

in the hospital wards, where rheumatism in its severer forms is most frequently encountered.

The purpose of this paper is simply to state my observations, not with any idea of finality, but rather to attract attention to a peculiar discrepancy, in the hope that it may lead to a renewal of interest. If it will stimulate a more productive search for these nodules in this country, so that it can be established by widely distributed observers whether the rheumatic nodule is a more or less definite quantitative accompaniment of rheumatism, or whether it has a very selective geographic habitat, its purpose will have been amply fulfilled.

I am, perhaps unfortunately, not in a position to give definite statistical data as to the incidence of these nodules. Such data would, however, I believe, rather detract from the more important observation that I wish here to emphasize, that these nodules do not occur in certain stages and types of rheumatism; that they do occur fairly frequently in others, and that in certain types they are the rule.

A brief review of the early literature on this condition is of interest per se, but even more so because it illustrates how quickly we often see a large number of cases when once our attention is attracted forcibly to a phenomenon that was hitherto unknown, or considered too rare to be of interest.

The first recorded case of rheumatic nodules was that of Meynet.[9] He saw this case in 1874. The patient was a boy, 14 years of age, in whom the nodules developed while he was being treated at the hospital. Rehn[10] quotes Meynet and adds a second case in a child with recurring rheumatism. He later reported another case. Hirschsprung[11] of Copenhagen reported four cases. Troisier and Brocq[12] reported a case in an adult. Barlow and Warner[13] saw their first case in 1875, and in 1881 they reported twenty-seven cases, the patients being between the ages of 4½ and 18 years. But little has been added to our knowledge of these cases since the publication of this brief but comprehensive paper. Twenty-five of the twenty-seven cases had definite symptoms of rheumatism. Twenty-six had definite cardiac involvement and the remaining case had a peculiar heart with a "murmurish first sound." Eight patients died of heart disease. Ten had chorea at the time the nodules were observed; one had had

9. Meynet: Lyon Med., No. 49, 1875. Quoted from Barlow and Warner.
10. Rehn: Gerhardt's Handb. **3**:21, 1878.
11. Hirschsprung: Arch. f. Kinderh. (March) 1881. Quoted from Barlow and Warner.
12. Troisier and Brocq: Rev. de méd. (April) 1881. Quoted from Barlow and Warner.
13. Barlow and Warner: Trans. Seventh Internat. Med. Congress, London **4**:116 (Aug.) 1881.

chorea some time before. They described the morphology, localization and duration of the nodules; established their pathognomonic relationship not only to rheumatism, but also to endocarditis; and pointed out on the basis of three microscopic examinations, the close structural relationship of the nodules to the vegetations on the valves in an endocarditis.

Since this time there has been a sort of stereotyped uniformity about the literature of these cases confined chiefly to textbooks. British writers like Cheedle, Poynton, Hutchinson, Thompson, Still, Cautley and others, all emphasize their relative frequency, their association with heart disease, their grave significance when abundant and recurring, their structural identity with "submiliary nodules" of the heart with some difference of opinion as to the exact histogenesis.

The status of German opinion is shown in the fact that Heubner[14] does not mention them; in Pfaundler's undisguised elation on seeing a rare case, in this instance so manifest that it could not escape detection; and in Ibrahim's[15] naive statement in Pfaundler and Schlossman (1906): "A special form of this disease, almost wholly peculiar to childhood, is rheumatismus nodosus, first described by Reynet in 1875 and popularized through Rehn and Hirschsprung. Over forty cases in children have been described up to this time (chiefly by English authors)."

Our own literature has been quoted sufficiently.

The rheumatic nodules vary in size from that of a pinhead to that of a filbert in our experience; commonly, they are the size of a split pea. Doubtless they are often beyond the reach of the eye or the finger. They are always subcutaneous, never reddened or attached to the skin, and rarely tender. When the skin is loose over them there is no discolorization; if taut they appear whitish or grayish and shiny. The small ones have well been likened to a boiled sago grain. Usually they are discrete, roundish or hemispherical, with symmetrical outlines, but sometimes they are clustered, or elongated, or even radiate out in different directions. Sometimes they can be felt better than seen, but this is commonly not the case if the joint about which they occur is properly flexed, or if the skin is otherwise lightly drawn over them. On the head they must always be palpated. Over the patellae they are often better seen than felt.

They are usually distinctly movable, in fact, the smaller ones about the deeper structures of the elbow and sometimes elsewhere would often be hard to distinguish except for that fact. On the periosteal

14. Heubner: Lehrbuch f. Kinderh., 1906.

15. Ibrahim, J.: Acute Articular Rheumatism. Pfaundler and Schlossmann, Handb. der Kinderh. **1**: Part 2, 881, 1906.

surfaces, such as the patellae and the spines of the vertebrae, they are often more firmly anchored and sometimes seem to be quite immovable.

They vary in number from one or two to a hundred or more — commonly there are eight or ten. If numerous, they are always widely scattered. The greater the number the more surely will some of them be very large, especially over the patellae, the back of the head, or on the spines of the vertebrae.

They occur always in connection with the deep fascia, the tendons, or the periosteum, but they have favorite localizations. Thus they are nearly always found, if present at all, about the condyles of the humerus, and in and about the olecranon process. Occasionally, a very large one is found near the end of the olecranon process that is almost indistinguishable till it is found to be movable. It is especially about the elbow that the joint must be flexed properly, and careful search made both with the eye and the finger. They are rarely as large here, except over the olecranon, as they are in other locations where they are connected with the periosteum. In our experience they occur relatively frequently and very large, not only along the margins as commonly reported, but over nearly the whole of the patella commonly on both knees. When the knee is flexed sharply they are very conspicuous. They are frequently found over the malleoli, especially over the external malleolus, on the styloid processes of the radius and of the ulna, but they are never large or numerous here. A striking and not uncommon location is on the knuckles, and sometimes the tendon of nearly every knuckle is the seat of from one to five nodules. If the hand is open they quite escape notice; if the hand is tightly clenched they appear as prominent shining, whitish or grayish clusters along the tendons. Less frequently they occur on the toes.

In six or eight cases the most prominent nodules have been under the scalp, always on the back of the head. The hand should always be passed over this region. If present, there are usually from two to six, or more, large bumps nearly the size of a hazel nut.

Occasionally they show a distinct predilection for the spine, where they may be hard to find if laterally or deeply seated, and most conspicuous if located on the tips of the spinous processes. In a recent case nearly every spinous process for some distance was crowned by a large nodule and one of unusual size in the region of the first dorsal vertebra had been manipulated actively by a nonmedical practitioner in an effort to reduce a "dislocation" that was causing his "heart disease."

Occasionally they occur along the spines and vertebral borders of the scapulae, less frequently about the shoulder joint and along the crests and articulations of the ilia. I have never seen them along the

tendons distant from a joint, or in the palm of the hand, or about the sternum, though they have been reported in these places.

Rheumatic nodules have a tendency to appear in groups, or waves. The life of the individual nodule varies from a few days to as many months.

It is not difficult to see why these nodules may be overlooked. One is traditionally sparing in manipulating a rheumatic patient, and especially in bending the joints. The elbows, hands and knees are commonly extended, and unless flexed they show nothing. The back of the elbow, too, is naturally turned away from the examiner in the bed patient, and it requires considerable movement to bring it into view and to palpate it properly. This is even more true of the spine of the bed patient. The spine, moreover, is naturally "bumpy." Passing the hand over the back of the head is easily omitted and in other locations the nodules must be looked for especially, and they are often hard to find. When the nodules are large and numerous they, of course, obtrude themselves.

On two points practically all observers are agreed, the rheumatic nodule means rheumatism, and it practically always means endocarditis. Its similarity in structure with that of valvular nodules would lead one to think that both are more or less simultaneous effects of an identical cause.

In the ordinary case of articular rheumatism or of chorea, without heart involvement, the rheumatic nodule does not occur. In those cases of cardiac rheumatism in which nodules do appear they commonly follow the heart involvement after some days or weeks. Usually, only in the severe cases do they appear at the same time or very rarely a very short time before the heart is evidently affected. One of my cases, a boy, six years of age, showed on my first visit an abundance of nodules, but I could not make out a definite endocarditis until the next day. He rapidly developed a severe and protracted endocarditis and pericarditis with many nodules throughout the course of the illness. The question of how early an endocarditis is recognizable clinically would here seem pertinent.

The ordinary endocarditis that one sees for the first time in the course of an acute articular rheumatism, or of a chorea, that quickly runs a favorable course under proper treatment is usually not accompanied by the appearance of nodules. The case that at once strikes one as a severe infection, and especially one complicated by pericarditis, shows them much more frequently. The same is true of recurring and persistent cases of rheumatic infection with renewed cardiac invasions even though the individual attacks may not seem very severe. While it is true, in general, that severity and chronicity of infection

are the most important factors in the production of rheumatic nodules, it is also true that nodules may occur in any case of cardiac rheumatism, and that they may be wholly absent in just those cases where experience leads one to expect them.

There is, however, a type of rheumatism in which we have nearly always found them, and usually abundantly. These are the cases in which there is severe endocarditis of long standing, often accompanied by pericarditis, where the process is active and progressive in spite of all treatment; where there is a low but nearly constant temperature; where there is wasting, with or without anasarca, and a yellowish, gray, cachectic color. These cases that sway between life and death for weeks and months, not alone because of a broken compensation, but also even more because of a smoldering, uncontrollable infection that ever and anon lights up again, are in our experience rarely free from a large number of nodules. It is in these cases that a new and abundant crop of nodules gives an ominous warning that there is renewed invasion of a serious nature.

While the number and the size of the nodules in a general way run a parallel course with the severity of the rheumatic infection, this is not always the case; nor, from my experience, can I agree with Cheedle[16] when he says: "In the acute rheumatic fever of children when the nodules are many and large they have an ominous association with progressive endocardtis and pericarditis of the most serious kind; they indicate a grave danger, a carditis which is uncontrollable and advances almost invariably to a fatal termination." A number of our own cases that have had nodules as large and numerous as I have ever seen have ultimately, though nearly always very slowly, run a favorable course and have been left with only a chronic valvular lesion.

Though we ordinarily think of the presence of rheumatic nodules as an indication that the rheumatic infection is still active, it seems, however, often to be at least slumbering. The appearance of new nodules is here of far greater significance than the long persistence of old ones. Individual nodules will often persist for weeks and even months after all acute symptoms have subsided. We had at one time two children in the ward, each convalescing from severe cardiac rheumatism, between whom there was a very active daily rivalry accompanied with much hilarity as to which still showed the largest number and size of an old crop of nodules. In a case now under Dr. Krost's care, the child was up and about while he still had well marked nodules, though with us the rest in bed in these cases is one of months rather than of weeks.

Since rheumatic nodules are considered pathognomonic of rheumatism, it is evident that they are, at times, of considerable diagnostic

16. Cheedle: Quoted by M. H. Bass.

value. The pathognomonic symptom is always cherished. Rheumatism in children where articular symptoms sink to a minimum and cardiac manifestatons rise to a maximum has always presented difficulties in diagnosis. Parenthetically, I might say that these difficulties are rather increased by the present tendency to make it one of a group rather than an individual and the nodules will help to maintain for rheumatism that individuality that the pediatrician will long hesitate to deny it. Several years ago I saw a child with very high fever, delirium, very stiff and painful neck, in whom I suspected a meningitis until I found on more careful examination that he had a vigorous crop of nodules about the elbows. A definite endocarditis on the following day and a subsequent pericarditis confirmed the diagnostic value of the nodules in enabling me to make a diagnosis of rheumatism on the first day. Within the last week I saw a boy, 10 years of age, with a chronic infection who for two months had worn a cast over a supposed tubercular knee. He had now dullness and distant breath sounds over the left base, an endocarditis and probably a pericarditis and a very stiff and painful and rigid neck and back with a marked and persistent forward bend and with no other active joint symptoms. The presence of nodules made it more than probable that the whole process was rheumatic and not tubercular.

A peculiar interest has, to my mind, always attached to the nodules found on the knuckles. The hand is the only exposed part in this connection. By a glance at the knuckles in such a case we have been able to say: "This child has rheumatism; it is probably active; it is severe; he has an endocarditis."

Not the least value of the rheumatic nodule has been its contribution to the view that chorea is rheumatism.

A final word as to the geographic incidence of rheumatic nodules. Is it probable, as suggested by Dunn,[5] that they "are probably seen more often in some parts of this country than in others," say in Chicago as compared with Boston, or New York? A priori this seems improbable. It is difficult to see any determining climatologic or meteorologic or racial differences in the two localities. I might again refer to what I said five or six years ago which quite parallels the current statements of our textbooks. Finally, I might quote Cotton[17] who practiced pediatrics in Chicago for forty years. He says: "The fibrous nodules infrequently encountered in this country are common in England."

30 North Michigan Boulevard.

17. Cotton, A. C.: Diseases of Infancy and Childhood, 1906, Philadelphia, J. B. Lippincott & Co., p. 630.

CEREBRAL COMPLICATIONS IN MUMPS*

HORTON R. CASPARIS, M.D.
BALTIMORE

Involvement of the central nervous system during the course of mumps has been recognized for many years. In 1761, Hamilton[1] reported a case in a young man who died on the second day of cerebral manifestations which had developed during an attack of mumps. Frenel,[2] in 1812; Malabouche,[3] in 1867; Niemeyer,[4] in 1869, and others reported single instances of this complication. Then in 1877 Gailhard[5] reported six cases. He described in more detail the clinical manifestations, but it was not until 1880 when Maximovitch[6] at necropsy found the surface of the brain edematous, the sulci filled with semifibrinous exudation and hyperemia of the pons and of the spinal cord, that there was any suggestion of the actual nature of the involvement of the central nervous system. Since then many cases have been reported, especally by the French, but it is only recently that a systematic study of the condition has been made by modern clinical and laboratory methods.

In contrast to the sixty or more cases found in the French literature, in this country as in the other countries, only a few scattering cases have been reported. In 1851 Lindsly[7] saw two deaths in adults two days after the onset of a meningitis which came on during the course of mumps. Bromwell[8] reported two cases in 1899, Woodward[9] one case in 1904, Acker[10] two cases in 1914, Kaunitz[11] three cases in 1918, Larkin[12] two cases in 1919, and we have seen the following two

---

* From the Harriet Lane Home, Johns Hopkins Hospital, and the Department of Pediatrics, Johns Hopkins University.

1. Hamilton: London M. J. **19.**
2. Frenel: Th. de Strasbourg, 1812.
3. Malabouche: Th. de Paris, 1867.
4. Niemeyer: Traite de pathologie interne, 1869.
5. Gailhard: Th. de Montpellier, 1877.
6. Maximovitch: St. Petersb. Med. Wchnschr., p. 185, 1880.
7. Lindsly: Va. M. Gaz., 1851.
8. Bromwell: Am. J. Obst. **40**:383, 1899.
9. Woodward: The Ocular Complications of Mumps, N. Y. M. J. **79**:20, 1904.
10. Acker: Parotitis Complicated with Meningitis, Am. J. Dis. Child. **5**: 399 (May) 1913.
11. Kaunitz: Mumps Meningitis, J. A. M. A. **70**:1448 (May 18) 1918.
12. Larkin: Mumps-Meningitis; Report of Two Cases with Necropsy Findings, Mil Surg. **44**:92, 1919.

cases in Dr. Howland's service in the Harriet Lane Home of the Johns Hopkins Hospital. They will be given somewhat in detail, for they present fairly typically the symptoms and course of the disease.

CASE 1.—The patient, 7 years of age, was admitted to the hospital Feb. 25, 1914, with convulsions. Three days before admission he had a chill and complained of headache. The following day he became delirious and vomited "almost continually." On the morning of the third day (the day of admission) he had a convulsion which lasted about ten minutes.

When admitted to the ward the patient was delirious, was very hyperesthetic and screamed whenever touched. At times he appeared to be in pain, would flex his thighs on his abdomen, move his head from side to side and fling his arms about. Finally, after a severe attack of delirium he became semistuporous after which he could be roused and would answer in a disoriented way. The hyperesthesia disappeared at this time. The neck was rigid, the knee kicks were exaggerated and Kernig's sign was positive. Neurologic examination was otherwise negative. The eyegrounds were negative. The temperature was 102 F. and the pulse 104. The leukocytes numbered 15,000 with a normal differential count. The von Pirquet test was positive. Lumbar puncture yielded a clear fluid under increased pressure with 470 cells per cubic millimeter, mainly lymphocytes. The test for globulin was negative at first but subsequently became weakly positive. Cultures on agar and blood agar plates gave no growth. The spinal fluid Wassermann and the colloidal gold test were both negative.

After the patient had been on the ward about eighteen hours there was noted a small bilateral swelling in the region of the parotid glands which was more marked on the left side than on the right. The submaxillary and sublingual glands were not involved. Subsequently the swelling in the parotids became pronounced and the submaxillary glands became involved but the patient's condition after admission rapidly improved and by the third day (sixth day of disease) he was feeling well. On that day the parotid swellings had begun to recede. The positive Kernig's sign, the tâche cerebràle, and the positive spinal fluid findings persisted for some days.

CASE 2.—The patient, 8½ years of age, was admitted to the hospital July 19, 1918, with headache and fever. Four days prior to admission he waked up in the night with the above symptoms, and during the next day he vomited two or three times. The headache and fever continued with constipation as practically the only symptoms until the night before admission when the patient became slightly delirious.

On admission to the ward he answered questions readily, was fairly alert, and did not look particularly ill. There was a tâche cerebràle, the reflexes were normal, there was no hyperesthesia, and the neck was not rigid, but there was a rather markedly positive Kernig's sign. The eye grounds were normal. The temperature was 102 F. and the pulse was 96. The leukocytes numbered 6,200 with a normal differential count. The von Pirquet test was positive. Lumbar puncture yielded a clear fluid under increased pressure with 900 cells per cubic millimeter of the small lymphocytic type. The test for globulin was positive. Cultures on blood agar and ascitic fluid agar gave no growth.

During the first day in the hospital small but definite swelling in the region of the right parotid became evident. The next morning the swelling was pronounced and subsequently the left submaxillary and also the submental glands became involved. After the lumbar puncture which was done on admission to the ward the meningeal symptoms rapidly disappeared. The mumps, which was just beginning, ran its course rapidly and within five or

six days the patient was well. There was a persistence of the positive Kernig's sign and the positive spinal fluid findings. These, however, gradually became less marked.

The occurrence of frank symptoms of central nervous system involvement as a complication of mumps is relatively rare. It seems to vary with different epidemics, however, and occurs more often in epidemics than in the ordinary run of hospital cases. This is evident from the reports from army camps especially in France. Of the cases reported the complication has occurred more frequently in males than in females. The onset comes more often at the height of the mumps. The time varies, however, as it might appear later and in a few cases it has preceded the parotid swelling as seen in our two cases also in single cases reported by Voisin,[13] Colomb and Mercier,[14] and Cayrel. The first symptoms usually are a rise in temperature, headache and vomiting, but these are not constant and the symptoms may be such as to be attributed to uncomplicated mumps. However, one does find headache, vomiting, drowsiness, rigid neck, or a positive Kernig's sign, something to suggest an involvement of the central nervous system.

Our first case presented the more severe train of symptoms. A chill marked the onset which was followed by a sharp rise in temperature, then nausea and vomiting, a very marked headache, convulsions, delirium and constipation. Coma may follow these with or without a fatal termination. The neck is usually rigid, although not always so. Kernig's sign is positive, and it frequently persists for some days after the symptoms have disappeared. As a rule, the disease is benign and runs a short course, ranging from two or three to about ten days. Occasionally, however, there are residuals of the involvement of the nervous system which may appear quite serious. Neither of our cases presented any of these, but the following are some that have been reported: Inequality of the pupils, failure of accommodation, strabismus, muscular twitchings of the face, unilateral facial paralysis, deafness.

Woodward[9] reported a case of ocular neuritis with marked impairment of vision. Sicard[15] reported a case of hydrocephalus; Lannois and Lamoine, a case of aphasia and right hemiplegia; Monro,[16] a case

13. Voisin: Soc. med. des hôp., Par., 1916.
14. Colomb and Mercier:: Méningisme ourlien, Soc. méd des hôp. **33**:240 (Feb.) 1912.
15. Sicard: Hydrocéphalie acquise par méningite ourlienne, Rev. neurol., Paris **27**:706, 1914.
16. Monro: Observations published by Healy, Lancet **2**:280, 1883.

of a man who suffered from unsteadiness of gait, incoordination of speech, and agraphia; Sorel,[17] a case of aphasia which persisted for fifteen months; Findley,[18] a case of infantile hemiplegia, and so on. These, however, have all been temporary, lasting a few days, weeks, or months, except the deafness which is usually permanent. A number of cases of permanent deafness have been observed in otherwise uncomplicated mumps, cases of temporary unilateral facial paralysis, and such conditions as nystagmus, herpes, polyneuritis, flaccid paraplegia and complete flaccid paralysis of all four limbs. The character of the spinal fluid was not mentioned in this last group of cases.

FINDINGS IN SPINAL FLUID

Day of Disease	Case 1	Case 2
Third	Cells 470; globulin negative	
Fourth	Cells 564; globulin negative	Cells 900; globulin positive
Sixth	Cells 290; globulin positive, free from symptoms	Cells 680; globulin positive
Ninth	Cells 193; globulin negative	Free from symptoms
Fifteenth	Cells 47; globulin negative	Cells 160; globulin positive
Twenty-ninth	.....	Cells 15; globulin positive
Thirty-sixth	Fluid normal	
Forty-first	.....	Fluid normal

The cerebrospinal fluid findings, although varying somewhat in degree, have been in general rather constant. The fluid has always been found sterile. It is clear and is usually under increased pressure at the first puncture. The test for globulin is usually positive, but not markedly so. It may be negative at first with a high cell count and later as the cell count decreases become positive, as is occasionally found in the spinal fluid of poliomyelitis. There is no film formation. The cells present are lymphocytes and the number varies greatly in different cases and with the stage of the disease at which the puncture is made. It ranges from slightly above normal (considering twelve or less per cubc millimeter a normal finding) to about two thousand. More frequently there are several hundred, although in one or two instances the fluid of the first puncture has given a normal count. It has been found that the number of cells bears no proportionate relation to the intensity of the symptoms. The return of the cellular increase to normal may require some time and continue for days or weeks after symptoms have disappeared. This was so in our cases (see table) but was even more marked in cases studied by Chauffard and Boidin.[18a] Concerning the type of cell in the spinal fluid, Dopter[19] did find in one case that there was a large number of

17. Sorel: Arch. de méd. mil., December, 1883.

18. Findley: Glasgow M. J. **65**:50, 1906.

18a. Chauffard and Boidin: Deux cas de méningite lymphocytique dans les oreillons, Soc. méd. des hôp. **21**:477, 1904.

19. Dopter: Soc. méd. des hôp., March 24, 1904.

polymorphonuclears as well as lymphocytes and in Lavergne's[20] case the polymorphonuclears and lymphocytes were about equal at first, but the former soon disappeared leaving practically all lymphocytes.

Further, in regard to the cerebrospinal fluid, the question has arisen as to whether or not the increase in cells is present only in the cases where there are frank manifestations of central nervous system involvement, or whether it also occurs in all cases of mumps. Monod[21] in 1902 was the first to make a systematic study with this point in view. He did lumbar punctures on eight children with mumps who presented no clinical evidences of meningeal irritation, and six out of the eight showed a definite increase of cells (lymphocytes) in the spinal fluid. On the other hand, Nobecourt[22] systematically punctured a number of cases of mumps, and he said that in none without meningeal symptoms did he get an increased cell count. Dopter[19] agrees with this statement that in simple mumps the spinal fluid is normal. Later, however, in 1917 de Massary, Tockmann, and Luce,[23] in an epidemic of mumps in the French army had sixteen definite cases of the so-called mumps meningitis. All of these presented the usual cellular increase in the cerebrospinal fluid. They went further and did lumbar punctures on forty other mumps cases which gave no evidence of meningeal irritation, and in most of them too there was the same type of cellular increase in the spinal fluid. This discrepancy might be explained by different interpretations of the clinical manifestations of the cases studied. In other words, there is already an acute infection present with its symptoms, and to say whether or not there is an involvement of the nervous system is not always easy, for in the milder cases the signs and symptoms of such may be equivocal. It certainly seems probable though from the above findings that involvement of the central nervous system in mumps is much more frequent than most of us have ever suspected.

The diagnosis is not difficult when accompanying the symptoms of central nervous system involvement there is a parotitis, and when one finds on lumbar puncture a clear spinal fluid with an increased cell count and without film formation. However, one has to be rather guarded in his diagnosis if the so-called meningitis occurs before the

---

20. Lavergne: Sur un cas de méningite ourlienne simulant une forme grave de méningite cérébro-spinal a méningocoques, Arch. méd. des Enf. **20**:182, 1917.

21. Monod: Reactions méningees chez l'enfant, Th. de Paris, 1902.

22. Nobécourt and Brélet: Méningite ourlienne avec lymphocytose cephalo-rachidienne, Soc. de pédiatric, **7**:282, 1905.

23. de Massary, Tockmann and Luce: La Méningite ourlienne, sa constance son évolution, sa durée, Bull. de l'Acad. de méd., Paris **78**:6.

appearance of mumps, as there are a number of other conditions in which one sees similar spinal fluid findings, the more familiar of these being tuberculous meningitis in the spinal fluid of which a film forms on standing, tubercle bacilli can usually be demonstrated, the globulin reaction is pronounced, and the cell count is usually lower (from 50 to 150); poliomyelitis, the spinal fluid of which may be very similar in that in both the globulin is usually weakly positive or may be absent at first with a high cell count then later become positive as the cell count decreases; syphilitic meningitis, the spinal fluid of which gives a positive Wassermann; and finally lethargic encephalitis, and lead meningitis and lead encephalopathy, the spinal fluids of all of these presenting nothing characteristically different from that of "mumps meningitis" except that in the former the cell count is usually much lower (from normal to 200). In the lead conditions the globulin reaction is more pronounced. The clinical manifestations usually aid though in differentiating these various types of meningitis.

As to the cause of the nervous complications of mumps, various theoretic explanations have been offered. Some of the older observers attributed the nervous symptoms to orchitis, others to alcoholism or hysteria; Eichorst,[24] to a passive cerebral congestion dependent on compression of the jugular vein by the parotid gland; Jaccoud,[25] to emboli from endocarditis caused by the parotitis infection; Lannois and Lemoine[26] who described the case of right hemiplegia and aphasia mentioned above felt that in all probability the lesion present was a diffuse meningo-encephalitis; a meningismus has been suggested; and finally Gallavardin[27] thought that it was a true meningitis due to the virus of mumps. He called the condition serous meningitis. Most of these explanations are readily eliminated.

As to the association with orchitis, the complication occurs in children before puberty during which period orchitis is very rare. Alcoholism of the parents and hysteria have been observed in only two or three cases. The "meningitis" appears occasionally before the parotid swelling as in our two cases and therefore it cannot be caused by cerebral congestion due to compression of the jugular vein by the parotid gland. Endocarditis has been associated with the so-called mumps meningitis in only one or two cases, and those were probably incidental. The spinal fluid findings rule out meningismus. Galla-

24. Eichorst: Ziemmsen's Handb., 1883.

25. Jaccoud: Brit. M. J. **2**:41, 1885.

26. Lannois and Lemoine: Des manifestations méningitiques des oreillons, Arch. de neurol. **11**:1, 1886.

27. Gallavardin: Complications nerveuses des oreillons, Gaz. des hôp. **71**: 1329, 1898.

vardin's[27] theory of the etiology then seems to be the most probable one, but as to the actual nature of the involvement of the central nervous system, there have not been enough fatalities to furnish satisfactory pathological data for this to be determined definitely.[28] However, considering the spinal fluid findings and the clinical manifestations, in my opinion, it seems more probable that there is an involvement of the encephalon as well as of the meninges, rather than an involvement of the meninges alone.[29]

---

28. Feiling: Mumps; a Critical Review, Quart. J. M., p. 255, 1914. Feisinger: Reunion médicale de la VIe armée méningites oreillons, Arch. de méd. et pharm. mil., Paris **66**:525, 1916. Roux: Les Méningites oreillons, Gaz. des hôp. **87.**

29. Thomas and Blackfan: Recurrent Meningitis Due to Lead. Am. J. Dis. Child. **8**:377 (Nov.) 1914. Voisin: Soc. méd. des hôp. d. Par., 1916.

A CASE OF DIABETES INSIPIDUS IN A GIRL OF TEN YEARS FROM THE STANDPOINT OF ENDOCRINAL INVOLVEMENT*

DAVID MURRAY COWIE, M.D.
ANN ARBOR, MICH.

Janet, T., aged 10 years, entered the pediatric service of the University Hospital, April 17, 1919, complaining of excessive appetite for water. Aside from an attack of measles when 6 years of age, and whooping cough at 7 years of age, she has always been well. Of late she has not been gaining normally in weight, and her appetite for food is poor. A careful consideration of the family and birth history, Wassermann and tuberculin (subcutaneous) tests enables us to rule out syphilis and tuberculosis as possible etiologic factors. There is no history of injury.

Two years ago, the mother noticed that the child passed an unusually large amount of urine, and that she was very thirsty. These symptoms have gradually increased so that now she frequently passes five or six quarts of urine daily. Since entering the hospital, the largest quantity of urine voided was 5,180 c.c. (5½ quarts); the largest intake of water 6,090 c.c. (more than 6 quarts). Ever since the child has complained of polydipsia she has had headache. These were infrequent at first, but now they occur three or four times a week and are associated with nausea and vomiting.

The following observations were made before and after the administration of pituitary extract:[1]

BEFORE AND AFTER ADMINISTRATION OF EPINEPHRIN

		Before Pituitary Extract		After Continuous Administration of Pituitary Extract	
	Water intake and output..	Table 1			
	Fixation of gravity test...	Table 2			
	Greatest variation	3		6	
	Phthalein excretion	70 per cent.			
April 26, 1919	Total chlorids, 24 hr. urine	8.9 gm.	May 12, 1919	11.5 gm.	
	Total chlorids, 24 hr. urine	10.18 gm.			
	Total ammonia	0.315 gm.		0.396 gm.	
	Total ammonia nitrogen...	0.261 gm.		0.480 gm.	Folin
	Creatinin	0.591 gm.	Weight, 50 lbs.	0.460 gm.	Folin
	Creatinin nitrogen	0.219 gm.		0.170 gm.	
	Creatin	0.0162 gm.		0.0145 gm.	Folin, Benedict, Meyers
	Blood urea	0.032 %		0.040 %	VanSlyke, Cullen
	Blood sugar	0.092 %		0.088 %	Benedict, Lewis
		0.086 %		0.091 %	

Examination.—Aside from a slight cervical adenitis and slight undernutrition, a carefully done, complete physical examination is entirely negative. The urine was of low specific gravity (1.001) and a large quantity was voided; otherwise it was negative. Blood examination showed: hemoglobin, 82 per

* From the Department of Pediatrics and Contagious Diseases, University of Michigan Hospital.

1. The same conditions as regards food, etc., were maintained during these observations; 0.75 c.c. surgical pituitary extract was used for the daily injection.

cent.; red blood cells, 4,550,000; white blood cells, 7,800, made up of polymorphonuclears, 63 per cent.; small lymphocytes, 32 per cent.; large lymphocytes, 4 per cent.; eosinophils, 1 per cent. The spinal fluid was clear and contained six cells per cubic centimeter. The phenol, Nona Apfelt and albumin tests were negative. Fehling's test was positive.

A roentgenogram of the head showed that the skull cap was thin with the usual markings; the coronal suture was not united; the anterior fossa was normal; the sella turcica was small, of the infantile type and, probably, physiologic; the sphenoid was not yet excavated. This examination was, therefore, negative.

TABLE 1.—Water Intake and Urine Output

Date	Water Intake Unrestricted, C.c.	Urine Output			Remarks
		Day, C.c.	Night, C.c.	Total, C.c.	
4/18/19	3,420	2,040	1,200	3,240	
4/19/19	1,950	750	1,380	2,130	
4/20/19	2,400	1,503	1,005	2,508	
4/21/19	3,780	1,800	1,560	3,360	
4/22/19	2,495	2,015	1,345	3,360	
4/23/19	4,800	2,760	2,505	5,265	
4/24/19	3,855	2,010	1,250	3,260	
4/25/19	4,110	2,850	1,140	3,990	
4/26/19	4,530	2,410	2,340	4,750	Blood and urine observations. Fasting since supper last night. 1 c.c. epinephrin, 1:1,000 at 2 p. m.
4/27/18	4,710	4,470	1,170	5,640	
4/28/19	4,890	3,570	3,420	6,990	95 gm. glucose. Blood samples taken
4/29/19	4,680	3,870	2,220	6,090	
4/30/19	5,880	3,270	3,180	6,450	
5/ 1/19	3,240	2,740	1,670	4,410	
5/ 2/19	5,540	2,250	3,180	5,430	
5/ 3/19	4,890	2,940	3,200	6,140	95 gm. glucose. Blood samples taken
5/ 4/19	4,570	2,730	2,200	4,930	
5/ 5/19	4,600	3,450	2,000	5,450	
5/ 6/19	2,220	570	1,700	2,270	0.75 c.c. surgical pituitary extract at 8:30 a. m.
5/ 7/19	1,380	1,420	250	1,670	0.75 c.c. surgical pituitary extract at 9:00 a. m.
5/ 8/19	1,290	450	400	850	0.75 c.c. surgical pituitary extract at 9:00 a. m. and 12 midnight
5/ 9/19	1,140	360	400	760	7 c.c. epinephrin at 8:35. Blood samples taken
5/10/19	2,310	1,380	550	1,930	0.75 c.c. surgical pituitary extract at 8:30 a. m.
5/11/19	1,590	1,465	360	1,825	0.75 c.c. surgical pituitary extract at 8:30 a. m. and 12 midnight
5/12/19	.....	.....	400	400	100 gm. glucose at 6:30 a. m. Observations on blood and urine. Fasting since supper last night
5/13/19	.....	.....	400	.....	0.75 c.c. surgical pituitary extract at 10 a. m. Discharged
5/14/19	.....	.....	.....	3,840	
5/15/19	4,500	.....	.....	5,280	
5/16/19	1,980	.....	.....	1,800	
5/17/19	2,340	.....	.....	2,280	
5/18/19	2,430	.....	.....	2,520	
5/20/19	5,030	.....	.....	5,530	

With a view to determining whether endocrinal involvement is present in this case, roentgenographic examination of the sella turcica was made, and epinephrin and glucose blood sugar curves were studied. It has been shown that, in certain diseases of endocrinal origin and in conditions in which certain of the endocrinal glands are involved, prolonged blood sugar curves are induced by the intramuscular injection of adrenalin and by the ingestion of glucose.

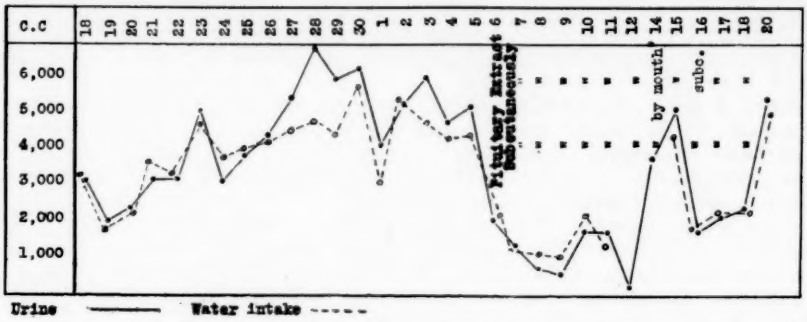

Chart 1.—Showing the water intake and urine output before and after the subcutaneous administration of pituitary extract.

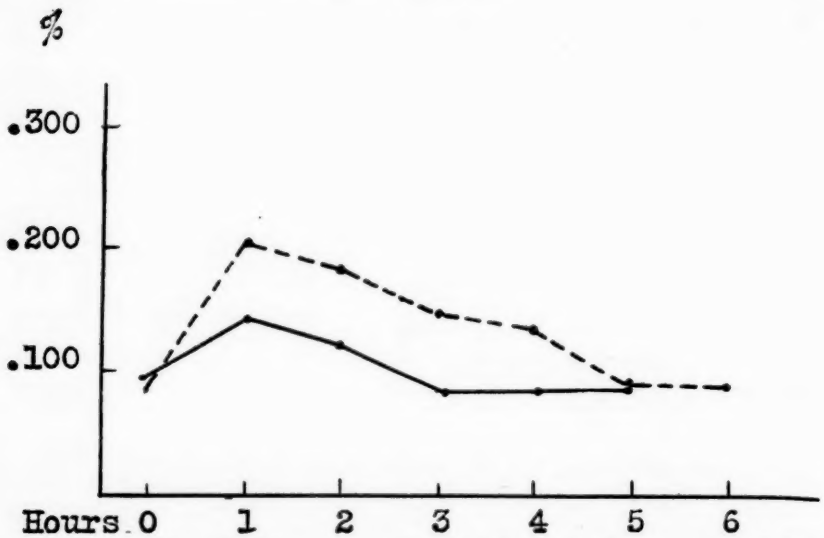

Chart 2.—The solid line shows the epinephrin blood sugar curve before pituitary extract treatment; the dotted line represents the curve after three days of pituitary extract treatment.

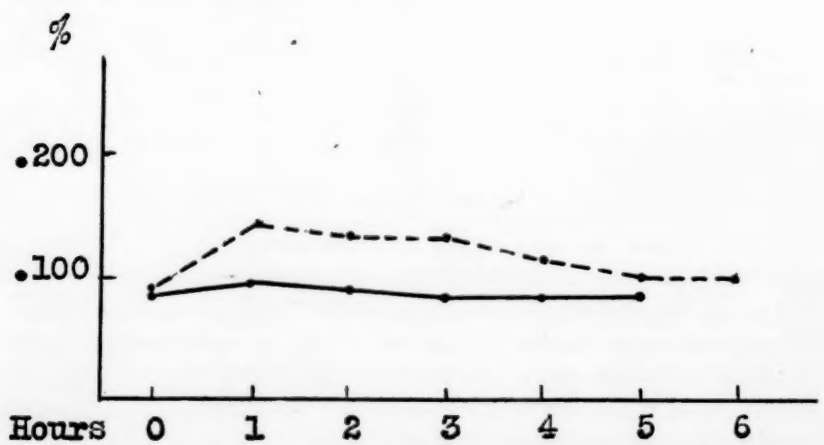

Chart 3.—The solid line shows the blood sugar curve before the pituitary extract treatment; the dotted line represents the curve after six days of pituitary extract treatment (See Table 4).

An examination of Tables 3 and 4 and of Charts 2 and 3 will disclose the fact that before the pituitary extract treatment was begun in this child the increase in the blood sugar induced by epinephrin follows the normal curve, that is, an abrupt rise which returns to normal before the third hour after injection, and that no change whatsoever was induced by the ingestion of glucose. The examination of the roent-

TABLE 2.—Fixation of Gravity Tests

Hours	Control, April 22, 1919			During Pituitary Extract Administration, May 11			Control, May 20		
	Water Intake, C.c.	Urine Output, C.c.	Sp. Gr.	Water Intake, C.c.	Urine Output, C.c.	Sp. Gr.	Water Intake, C.c.	Urine Output, C.c.	Sp. Gr.
7- 9 a. m.	330	325	1.001	480	360	1.001	360	720	1.001
9-11 a. m.	660	400	1.001	480	90	1.013	420	660	1.003
11- 1 p. m.	720	595	1.002	90	270	1.006	750	480	1.002
1- 3 p. m.	330	570	1.001	180	135	1.005	480	660	1.002
3- 5 p. m.	240	200	1.004	180	210	1.004	690	960	1.004
5- 7 p. m.	180	400	1.001	180	320	1.004	630	420	1.002
Total day	2,460	2,490	.....	.....	1,825	.....	.....	.....	.....
7 p. m.-7 a. m.	2,100	2,505	1.002	420	430	1.007	1,680	1,630	1.003
Total 24 hours	4,560	4,995	.....	.....	2,255	.....	5,050	5,530	.....

TABLE 3.—Changes in Blood Sugar Following Subcutaneous Injection of 1 C.c. Epinephrin

	Before Pituitary Extract Treatment Was Begun, April 26, 1919; per Cent.	After Continuous Administration of Pituitary Extract, May 9, 1919; per Cent.
Control	0.092	0.088
1st hour	0.143	0.204
2d hour	0.128	0.189
3d hour	0.088	0.149
4th hour	0.089	0.137
5th hour	0.089	0.088
6th hour	.....	0.089

TABLE 4.—Changes in Blood Sugar Following Ingestion of Glucose, 2.7 Gm. Per Pound Body Weight

	Before Pituitary Extract Treatment Was Begun, Weight 50 lbs., April 28, 1919; per Cent.	After Continuous Administration of Pituitary Extract, Weight 52 lbs., May 12, 1919; per Cent.
Control	0.086	0.091
1st hour	0.098	0.143
2d hour	0.090	0.134
3d hour	0.084	0.134
4th hour	0.089	0.114
5th hour	0.089	0.101
6th hour	.....	0.090

genogram must also be considered negative. Hence, from these observations, one can only conclude that there is no evidence of endocrinal involvement in this case, and that it should be placed in the group of idiopathic diabetes insipidis.

It is of interest to note that, during pituitary treatment, the blood sugar curves show the characteristics of endocrinal disturbance. We have, thus, induced an artificial hyperpituitarism.

OBSERVATIONS ON THE TOXIC PROPERTIES OF HEATED AND DECOMPOSED MILK AND OF MILK CULTURES OF *BACTERIUM WELCHII* (*BACILLUS AEROGENES CAPSULATUS*)*

WILLIAM W. FORD, M.D.
BALTIMORE

It has been previously shown by Ford and Pryor[1] that milk heated to temperatures of from 80 to 85 C. for from twenty to thirty minutes undergoes a violent fermentation with the production of acids and the evolutions of gas when incubated at 37 C. This change is due primarily to the constant presence in Baltimore milk of the spores of the gas bacillus (*Bacterium Welchii*), but in addition to this organism other spore bearing anaerobes and a great variety of spore bearing aerobes are also present in milk thus treated. The anaerobes in market milk were described several years ago by H. R. Brown[2] working in the laboratory of Prof. Theobald Smith at Harvard Medical School, and recently the aerobic spore bearing bacteria present in milk were investigated by Lawrence and Ford.[3] In the further study of the fundamental changes which may take place in pasteurized milk it has been necessary to investigate the properties of heated milk after decomposition has set in, and attention has already been called by Ford and Lawrence[4] to the presence of hemolytic substances of bacterial origin in such materials. The whey from samples of Baltimore milk heated to 80 C. and incubated at 37 C. also contains poisonous substances. Thus, May 22, 1918, six samples of market milk were heated to 80 C. for twenty minutes and incubated at 37 C. Of these, five showed the characteristic reaction of "stormy fermentation." These samples were filtered free from clot, neutralized with KOH and then filtered through

*From the Department of Bacteriology, School of Hygiene and Public Health, Johns Hopkins University.

1. Ford, W. W., and Pryor, J. C.: Observations on the Bacteria Found in Milk Heated to Various Temperatures, Bull. Johns Hopkins Hosp. **25**:270 (Sept.) 1914.

2. Brown, Herbert R.: A Study of Some of the Spore Bearing Anaerobic Bacteria in Market Milk, Forty-First Ann. Rep. Mass. State Board of Health, 1910.

3. Lawrence, J. S., and Ford, W. W.: Spore Bearing Bacteria in Milk, J. Bacteriol. **1**:277 (May) 1916.

4. Ford, William W., and Lawrence, Joseph H.: Hemolytic Substances in Heated Milk and in Milk Cultures of *B. welchii*, Bull. Johns Hopkins Hosp. **28**:245 (Aug.) 1917.

filter paper. The resulting filtrates, turbid and full of bacteria, were tested by subcutaneous inoculation on animals with the results as shown in Table 1.

TABLE 1.—TOXICITY OF NEUTRALIZED WHEY FROM HEATED AND DECOMPOSED MILK

No. of Sample	Weight of Guinea-Pig, Gm.	Dose, C.c.	Result
1.	450	5	Death in 24 hours
	370	3	Death in 24 hours
2.	400	5	Death in 24 hours
	370	3	Death in 72 hours
3.	300	5	Death in 24 hours
	320	3	Death in 48 hours
4.	210	5	Death in 24 hours
	220	3	Death in 24 hours
5.	200	3	Death in 24 hours
	120	1	Death in 24 hours

From this table it may be seen that the neutralized whey from the samples of milk which have shown the violent decomposition is always poisonous to small animals, bringing on a fatal result in from twenty-four to seventy-two hours. The changes produced by the different samples of whey were practically identical. At the site of inoculation there was an intense edema and congestion with the presence of large quantities of gelatinous reddish fluid in a mass of necrotic tissue. This tissue was studded with gas bubbles and there were frequently large gas cysts in the adjacent inguinal regions. On microscopic examination the necrotic material showed a few intact blood corpuscles and evidently an active local destruction of blood had taken place. Many bacteria were present in the exudate, both gram-positive and gram-negative in staining properties, but usually the large elements characteristic of *Bacterium welchii* were in the majority. The spleen was enlarged and congested, the acute splenic tumor seen in infections. No special changes were noted in the other organs. The urine in the bladder was straw colored, not tinged with hemoglobin. The blood in the heart was usually clotted and on microscopic examination a few gram-positive bacteria with the morphology of the gas bacillus could occasionally be made out.

TOXICITY OF FILTRATES

The samples of whey used in these tests contained a great variety of bacteria so that the toxicity exhibited by them might be due to the organisms themselves or to the presence of poisonous substances produced by their multiplication. The material from the five samples previously employed was therefore passed through Berkefeld candles to obtain bacteria-free filtrates. The resulting fluid was clear, straw-colored, slightly viscid. When tested on animals it proved to be poisonous as shown in Table 2.

TABLE 2.—Toxicity of Bacteria-Free Filtrates from Heated and Decomposed Milk

No. of Sample	Weight of Guinea-Pig, Gm.	Dose, C.c.	Result
1.	400	10	Death in 24 hours
	360	5	Death in 24 hours
	320	2½	Recovery
	190	1	Death in 72 hours
2.	300	10	Death in 24 hours
	380	5	Recovery
3.	420	10	Death in 4 hours
	220	5	Death in 6 hours
	250	2½	Death in 3 days
	140	1	Death in 10 days
4.	370	10	Death in 6 hours
	350	5	Death in 6 hours
	450	3	Death in 15 days
	160	1	Death in 12 days
5.	350	10	Death in 4 hours
	240	5	Death in 6 hours
	330	2½	Death in 48 hours
	250	1	Death in 6 days

In the animals dying acutely in this series no changes were observable at necropsy beyond a congestion at the site of inoculation. In the animals surviving for from twenty-four to forty-eight hours there was much edema and congestion at the point of inoculation with the accumulation of considerable quantities of reddish fluid in the dependent parts. The internal organs showed no special changes. The urine in the bladder was straw colored, not tinged with hemoglobin. In the animals dying at the later periods the only macroscopic change was the wide area of necrosis at the site of inoculation, often so extensive as to lead to a considerable slough of the skin at this point.

From these observations it is evident that the whey from the samples of milk heated to 80 C. and then allowed to decompose contains a definite toxic principle which is either a protein decomposition product or a bacterial secretion. This milk toxin is thermolabile, being destroyed by exposure to a temperature of from 70 to 80 C. for thirty minutes. This is shown in Table 3.

TABLE 3.—Heat Destruction of Milk-Toxin, Sample 3

Weight of Guinea-Pig, Gm.	Dosage	Result
330	10 c.c. raw	Death in 24 hours
550	10 c.c. heated to 60 C. ½ hour	Death in 24 hours
360	10 c.c. heated to 70 C. ½ hour	Death in 24 hours
315	10 c.c. heated to 80 C. ½ hour	No effect

This experiment was repeated a number of times and in general the results were consistent. The toxic substance is always thermolabile and practically always destroyed at temperatures ranging from 70 to 80 C.

SOURCE OF MILK TOXIN

The poisonous action of the whey from these samples of decomposing milk cannot be attributed to the acids present in the milk since all the injections were made with carefully neutralized material. The

source of this milk toxin must, therefore, be looked for in some of the bacteria present in the milk which might give rise to the poison by their growth. The most common organism in Baltimore market milk is *Bacterium welchii,* and this species overgrows the others which may happen to be present in the milk, in the decomposition at 37 C. Experiments were, therefore, undertaken to determine whether the gas bacillus will produce substances in milk similar to those described above. Pure cultures were obtained from heated milk by the usual methods and large flasks containing 1,000 c.c. sterilized milk were inoculated by pouring into them the contents of a milk tube culture twenty-four hours old, about 15 c.c. After the usual stormy fermentation had appeared in from twenty-four to forty-eight hours the curd was removed by passing the material through coarse filter paper and the filtrate obtained after neutralization was tested on animals with the result shown in Table 4.

TABLE 4.—Toxicity of Neutralized Whey from a Twenty-Four Hour Culture of B. Welchii

Weight of Guinea-Pig, Gm.	Dose, C.c.	Result
350	2	Death in 24 hours
390	1	Death in 24 hours
295	½	Death in 24 hours

The changes found at necropsy were practically the same in the three animals. At the site of inoculation there was an edema and necrosis spreading out over a large area of the abdominal wall. The necrotic material was studded with small gas cysts while there were larger collections of gas in the adjacent inguinal regions and often between the layers of abdominal muscles. Microscopic examination showed but a few intact blood cells and gram-positive and gram-negative bacteria of the morphology of the gas bacillus in great profusion. The spleen was swollen and hyperemic but definite macroscopic lesions in the other organs were not noticeable. The urine in the bladder was straw colored, not tinged with hemoglobin. The blood in the heart was clotted, and showed no gas bubbles. Microscopic examination revealed the blood cells practically intact with an occasional gram-positive organism with the morphology of the gas bacillus enmeshed in the corpuscles.

From these observations it is evident that the changes seen in the animals inoculated with the pure culture of the gas bacillus are strikingly similar to those found in animals inoculated with the samples of decomposed milk. Other cultures of the gas bacillus were tested upon guinea-pigs and in all instances the material was definitely poisonous It was also poisonous to rabbits which succumbed regularly to abou 3 c.c. for a 1,500 gram animal.

TOXICITY OF FILTRATE FROM MILK CULTURES OF BACTERIUM WELCHII

The poisonous character of the milk culture of the gas bacillus may be due either to some pathogenic property of the organisms by virtue of which they may multiply in the tissues and bring on the death of the inoculated animal or to the production in the milk of some poisonous or toxic substance. That this organism produces substances of this character is shown by testing the bacteria free filtrate on animals. Thus four different massive milk cultures of the gas bacillus were neutralized, freed from the curd by passing through filter paper and then filtered through Berkefeld candles. The clear, viscid, straw colored fluid was tested on guinea-pigs with the results as shown in Table 5.

TABLE 5.—TOXICITY OF BACTERIA FREE FILTRATE FROM MILK CULTURES OF B. WELCHII

No. of Sample	Weight of Guinea-Pig, Gm.	Dose, C.c.	Result
E1	250	10	Death in 20 hours
	260	5	Death in 20 hours
	230	2½	Death in 20 hours
	280	1	Death in 20 hours
E2	270	10	Death in 48 hours
	210	5	No effect
E3	230	10	Death in 20 hours
	280	5	Death in 20 hours
	280	2½	Death in 20 hours
	250	1	No effect.
E4	260	10	Death in 48 hours
	220	5	Death in 24 hours
	190	2½	Death in 10 days
	240	1	No effect

From this table it is evident that the whey from milk cultures of the gas bacillus when filtered free from bacteria is highly toxic. There are great variations in the amount of toxin present in the samples. Thus in the sample marked E 1 an acute death was produced by 1 c.c., while in that marked E 2, it required 10 c.c. to bring on this effect. This difference may be accounted for by the activity of the various cultures or by the quantity used for seeding the flasks. An attempt was made, therefore, to determine at what period in the development of the gas bacillus the maximum toxicity was present in the same culture. Several large flasks were inoculated with the same strain and the same quantity of gas bacillus culture and the toxicity of twenty-four, forty-eight and ninety-six hour cultures was determined. The results are shown in Table 6.

From these experiments it is evident that there are great variations in the toxicity of the filtrate at different periods, and that the results of animal inoculation are somewhat confusing. In general, it may be

said, however, that the maximum toxicity of the milk culture is apparently reached at the end of about twenty-four hours of active multiplication after which there is a fairly definite deterioration.

TABLE 6.—Toxicity of Filtrates from Milk Cultures of B. Welchii At Different Periods of Development

Weight of Guinea-Pig, Gm.	Dose, C.c.	Result
Filtrate 24 hours old—		
160	5	Death in 24 hours
160	3	Death in 24 hours
260	2	Death in 48 hours
200	1	Death in 8 days
330	½	No effect
Filtrate 48 hours old—		
210	5	Death in 7 days
210	3	Death in 7 days
215	2	Death in 48 hours
380	1	No effect
210	½	Death in 11 days
Filtrate 96 hours old—		
275	6	No effect
270	4	Death in 7 days
360	2	No effect
240	1	Death in 3 days
180	½	Death in 24 hours*

* The animal given ½ c.c. of the filtrate from the 96 hours culture was in poor condition at the time of inoculation.

HEAT DESTRUCTION

The thermal death point of the toxic substance in milk cultures of the gas bacillus was tested by heating the filtrate to various temperatures and inoculating guinea-pigs. The results are shown in Table 7

TABLE 7.—Heat Destruction of Filtrate from Milk Culture of B. Welchii

Weight of Guinea-Pig, Gm.	Dosage	Result
260	10 c.c. raw	Death in 24 hours
215	10 c.c. heated to 60 C. ½ hour	No effect
270	10 c.c. heated to 70 C. ½ hour	No effect
315	10 c.c. heated to 80 C. ½ hour	No effect

The toxic substance in the gas bacillus filtrate was thus destroyed at a temperature between 60 and 70 C. in half an hour. This experiment was repeated a number of times, but the result was by no means constant. In several instances enough toxic substance was left over after heating to 70 C., or even to 80 C., to kill the animal. In general, however, all was destroyed at about 60 C.

DISCUSSION

From the evidence here presented it is evident that when milk previously heated to 80 or 85 C. for from twenty to thirty minutes, is allowed to decompose, it contains bacteria and their products which are capable of giving rise to an acute death in small animals such as rabbits and guinea-pigs on subcutaneous inoculation. This is a true intoxication since the bacterial products when freed from bacteria by filtration through Berkefeld filters are always poisonous and bring on

fairly definite changes in animals. Milk cultures of *Bacterium welchii* are also poisonous and this poisonous action is likewise due to a toxic or poisonous principle present in the milk. The fact that the gas bacillus is the predominant organism in heated market milk and the similarity in the action on animals of the heated milk and the milk culture of the gas bacillus make it a reasonable conclusion that the poisonous quality of the decomposed milk is to be attributed to the development in it of this species which is always present in spore form. The fact that the point of heat destruction of the milk toxin is somewhat higher than that of the gas bacillus toxin may be explained on the assumption that the market milk may contain other substances than those produced by the gas bacillus, either protein decomposition products or the secretory products of other bacteria, aerobic or anaerobic in character. It should, however, be indicated that the toxic substance present in milk cultures of *Bacterium welchii* is also at times thermostabile, and may resist a temperature of 70 or 80 C. The exact relationship of the toxic substance formed in milk cultures of this species to the toxic substance recently described by Bull and Pritchett,[5] and shown by them to act as an antigen is not clear. The filtrates from the milk cultures are powerfully hemolytic, and some of their poisonous action may be due to blood destruction. The pathologic picture is not that of a pure hemolytic intoxication, however, as evidenced by the lack of hemoglobinuria and, in many instances, the animals die acutely without signs of local blood destruction. It is possible that there are two distinct substances in the milk cultures: one, hemolytic and present in considerable quantity, and destroyed at about 60 C. in thirty minutes, and one, toxic, usually resisting a temperature of 60 C., but produced quite irregularly. The latter substance is probably identical with the gas bacillus toxin investigated so carefully by Bull and Pritchett. Experiments to clear up this point are now in progress.

CONCLUSION

Milk heated to 80 or 85 C. for from twenty to thirty minutes and kept at 37 C. decomposes with the elaboration of definite poisonous substances. Similar and possibly the same products are found in milk cultures of *Bacterium welchii* (*Bacillus aerogenes capsulatus*) with which organism milk is always infected. The demonstration of such materials in heated milk should be kept clearly in mind in the consideration of the problem of milk pasteurization; and, under all circumstances, milk heated to temperatures sufficient to destroy vegetative bacteria but not their spores should be kept at a low temperature to

5. Bull, Carroll G., and Pritchett, Ida W.: Toxin and Antitoxin of and Protective Inoculation Against *B. welchii*, J. Exper. M. **26**:119 (July) 1917.

prevent the development of these spores and should be used within a brief interval after the heating. Whether the bacterial products found in this decomposed milk have any poisonous action when taken into the digestive tract has not been determined, but in view of their presence in milk the practice of keeping pasteurized milk for any considerable period of time before using should be condemned strongly. Finally, the occurrence of toxic substances in heated milk arising from the development of spore bearing bacteria requires a rigid supervision of milk production before pasteurization to prevent the entrance of organisms of this character.

PROGRESS IN PEDIATRICS

REVIEW OF RECENT LITERATURE ON THE NEW-BORN

N. O. PEARCE, M.D.
Instructor in Pediatrics, University of Minnesota
CHILDREN'S CLINIC OF MINNEAPOLIS

HEMORRHAGE OF NEW-BORN

Nothing has appeared in the recent literature which throws any additional light on the etiology of hemorrhagic disturbances in the new-born. Some progress has been made in the study of the pathology resulting from this condition, more especially of hemorrhage into the brain substance. However, treatment of this condition has been so perfected that there is a marked reduction in the mortality. The giving of whole blood, either citrated or not citrated, under the skin, repeatedly in mild cases, or directly into the superior longitudinal sinus or some other convenient vein in the more severe cases, has produced consistently good results.

Hedren[138] found intracranial hemorrhage in about 9.28 per cent. of 700 infant cadavers. The hemorrhage was restricted to the meninges in nearly 84 per cent., and cerebral hemorrhage accompanied the meningeal hemorrhage in enough cases to bring the total to 90.7 per cent. Clinical and anatomic details of each case are given. Delivery had been spontaneous in fifty of sixty-five cases. He ascribes great causal importance to intrauterine disturbances in circulation and compression of the bones of the head in the birth passage. In only three cases was inherited syphilis unmistakably present. Injury from the birth trauma may resemble in every respect injury from certain external causes after birth, and they may occur with rapid and easy spontaneous delivery. He argues that some standard procedure should be formulated and enforced for the examination of the brain in medicolegal cases.

According to Lespinasse,[139] the hemorrhage of new-borns is caused by syphilis, infection, hypoplasia of the coagulating elements of the blood and asphyxia. Occasionally, pathologic lesions of syphilis are found or a positive blood culture. The onset is usually from the second

138. Hedren, G.: Intracranial Hemorrhage in the Newly Born, Svenska Läk. Sällsk. Handl. **44**:53 (March) 1918.

139. Lespinasse, V. D.: Hemorrhagic Diseases of New-born, Treatment of by Direct Transfusion of Blood, with Clinical Report of Fourteen Cases, J. A. M. A. **62**:1866 (June 13) 1914.

to the fifth day, except in syphilitic cases, in which the onset is usually delayed until from the seventh to the ninth day. All of his patients were transfused with rather large amounts of blood given slowly into the femoral vein. None of the patients died from hemorrhage.

Writing on the subject of gastro-intestinal hemorrhage of the new-born infant, Balard[140] says that the hemorrhage is caused by infection or mechanical influence. The mechanical injury almost always results from disturbances of circulation, combined with difficult respiration, causing a sudden hypertension, especially in the abdominal organs. There is a reflux toward the umbilical veins, which explains the frequency of hemorrhage there. If the ligature is strong enough, this wave is thrown back on the abdominal organ, producing intense passive congestion. As a result of a severe infection, there is a grave change in the blood formula of the new-born with failure of the blood to coagulate. The mechanical hemorrhages are early and isolated, and the prognosis benign. Infectious hemorrhages appear late, are multiple in location and incoercible, producing death of the infant.

Green[141] describes intracranial hemorrhage in the new-born as arising from trauma in either normal or operative delivery, or from an associated hemorrhagic diathesis. Bleeding is usually subdural, but may be intraventricular. Diagnosis sometimes is difficult, refusal to nurse, pallor and facial edema are some of the earlier symptoms, and may soon be confirmed by appearance of more classic signs. Lumbar puncture for diagnosis and therapeutic relief, when the hemorrhage is infratentorial and cranial puncture when the hemorrhage is over the cerebral convexity is advocated. Operative decompression may give relief if the other measures fail. Cases associated with hemorrhagic disease should be transfused before craniotomy. Appearances in these cases of hemorrhagic disease are sometimes very deceptive. Frequently a baby will nearly bleed to death into the stomach or bowel before any blood is passed from the rectum. One ounce of blood lost by the new-born is equal to a quart of hemorrhage in an adult.

In Shuman's[142] case of intracranial hemorrhage in a child six weeks of age, a blood clot of 1½ ounces was found at necropsy. The author believes the condition is much more common than is generally supposed, quoting a percentage of from two to thirteen of all postmortem examinations of new-borns, as showing some degree of intracranial

---

140. Balard, P.: Gastro-Intestinal Hemorrhage in the New-born, Bull. Soc. obst. et de Gyenc., Par. **3**:508 (June) 1914.

141. Green, R. M.: Intracranial Hemorrhage in New-born, Boston M. & S. J. **170**:682 (April 30) 1914.

142. Shuman, J. W.: Intracranial Hemorrhage in the Infant, with History and Necropsy Report of a Case, J. A. M. A. **63**:1760 (Nov. 14) 1914.

hemorrhage. The commonest lesion is subdural hemorrhage and this may not manifest itself for days, or even weeks, after birth. Convulsions during these early weeks may be due to traumatic lesions of the brain. The tendency to hemorrhage not uncommonly present, especially in syphilitic infants, is probably an important factor in many of these cases. Discussing the cause of convulsions in infants less than three months of age, Francioni says that many of these infants who have these early convulsions develop normally afterward. Some convulsions are due to traumatic conditions or circulatory disturbance during the birth process. In others symptoms of spasmophilia, syphilis or tuberculosis became manifest later. Whatever the etiology, the type of convulsions were alike in all cases. It is manifest further that digestive disturbances may aid in bringing on convulsions in children who have a tendency in this direction. Owing to the stage of the development of the nervous system, the period before the third month is the one of greatest frequency of convulsions.

Gastro-intestinal hemorrhage in the new-born may be caused by the presence of the duodenal or gastric ulcer. Nuzum[143] reports three such cases. The lesion as found at necropsy showed an ulcerated area with no thickening or induration at the edge, but a band of adhesions extending from the region of the ulcer in the duodenum to the gall-bladder and intestines. He emphasizes the fact that vomiting of blood or bloody stools in the new-born may be due to gastric or duodenal ulcer, rather than to melena neonatorum. The ulcers may occur in the child in utero, and are of thrombotic origin. The bacteria originate in the mother and pass through the placental circulation. Many instances of pyloric spasm, indigestion and marasmus are due to these ulcers.

In a very excellent paper on asphyxia and hemorrhage of the new-born, Manton[144] says that 19 per cent. of all cephalic born children enter the world more or less asphyxiated. Poppel estimates that the death rate of asphyxiated children within the first week of life is seven times as great as of those born unasphyxiated. The customary division of asphyxia is into livida and palada. In the former we have the cyanosed face and upper thorax, the strongly pulsating cord, tonicity of muscles, with the reflexes active. This condition is usually ephemeral. In asphyxia palada, the child is pale and relaxed, and resuscitation does not insure continuance of life. The most important and serious manifestations of neonatal asphyxia are those of cerebral

143. Nuzum, T. W.: Gastric and Duodenal Ulcer in the New-born, Wisconsin M. J. **15**:111 (Sept.) 1916.

144. Manton, W. P.: Asphyxia Neonatorum and Its Sequelae, New York State J. M. **14**:302, 1914.

pressure. When death results necropsy reveals small punctate hemorrhages into the meninges of the brain and cord. Some children survive this stage and the minute hemorrhages are absorbed without leaving a trace. On the other hand, cerebral pressure symptoms may mark a more extensive and serious involvement of the brain structures in most if not all of these cases. The manifestations are the result of intracranial hemorrhage of a considerable extent. The author does not believe that asphyxia neonatorum is often directly responsible for the cerebral paralysis of children, chiefly perhaps for the reason that if there is serious intracranial damage the infant does not often survive the initial stage of the disorder. A series of cases are reported.

Twilight sleep, asphyxia neonatorum and cerebral hemorrhage all played a part in the death of the infant in the case reported by Beifeld.[145] The child was born during twilight sleep anesthesia. Labor was short and normal. There was the usual degree of asphyxia present. At the end of twenty-four hours the child's respiration rose to 90 per minute, and a convulsion lasting one-half minute, involving the left half of the body exclusively, took place. A few hours later the child was found in a position of exaggerated opisthotonos. The anterior fontanel was not bulging, but was only 1 cm. in diameter, and there was premature ossification of the cranium. Spinal puncture was unproductive. Exploratory operation exposing the right motor cortex showed no evidence of bleeding. At necropsy the chief findings were hemorrhages situated in the pial covering of the base of the left lobe of the cerebellum and in the pericardium and pleura. Death was due to asphyxia neonatorum.

Lindberg[146] reports a case of a child dying at the age of six weeks from purulent meningitis, which developed after an intracranial hemorrhage at birth.

A very interesting review of the progressive simplification of the technic of transfusion in the new-born, with hemorrhagic disease, is presented by Green.[147] The first method was that described by Vincent in 1911, which involved entering the external jugular vein. Kimpton's method by means of glass cylinders has proved best for direct transfusion with citrated blood. The mortality of the disease has been reduced by the more advanced methods from 50 to 10 per cent. Our greatest present need is knowledge of the etiology of the disease, especially the syphilitic factor.

---

145. Beifeld, Albert H.: A Case of Convulsions in a New-born Baby, J. Mich. M. S. **14**:125, 1915.

146. Lindberg, G.: Intermeningeal Hemorrhage and Its Infection in Young Infants, Hygiea, Stockholm **78**:1089 (Aug. 15) 1916.

147. Green, Robert M.: Transfusion in the Treatment of Hemorrhagica Neonatorum, Boston M. & S. J. **171**:715 (Nov. 5) 1914.

Lewisohn[148] advocates the use of citrated blood from the mother to stop the bleeding in hemophilia neonatorum. One hundred c.c. of 0.2 per cent. of citrated blood is given by exposing a superficial vein in the arm, and introducing the blood through a fine cannula. The mother's blood can be used safely, without hemolytic tests, for the new-born, while that of the father or other relatives may cause hemolysis.

Cherry[149] and Langrock report a series of thirty-four experiments on the blood of new-borns and their mothers for the purpose of ascertaining whether, in cases of hemorrhagic disease of the new-born, all mothers could be used as donors for their infants. In the thirty-four tests no hemolysis or agglutination occurred. They estimate that from 60 to 75 c.c. of blood are approximately sufficient to promote clotting and to supply the cellular elements to replace those lost by hemorrhage.

Helmholz[150] says that the superior longitudinal sinus is by far the easiest place to obtain blood from the new-born. The location does not vary, the walls of the sinus are rigid and cannot be pushed to one side. Any harm that might result from an occasional puncture of brain substance is negligible. The sinus also lends itself readily to intravenous injection. He described his technic in detail and shows a head-holding apparatus to be used in this work.

Goldbloom[151] describes an apparatus for superior longitudinal sinus work, which consists of an ordinary needle with stilet, on which is mounted an adjustable metalic block. The large size of this block is an aid in maintaining position and prevents tearing of the walls of the sinus.

A syringe for the purpose of introducing blood into the veins of the new-born described by Zingher,[152] has been found by him to be very efficient. He cautions against overburdening the patient's circulation with too large an amount of blood. From one to two and one half ounces are sufficient in an infant up to six months of age, three to four ounces for an infant from six months to one year of age and from four to six ounces for infants from two to four years of age.

---

148. Lewisohn, R.: Blood Transfusion in Hemophilia Neonatorum, Am. J. Obst. **77**:933 (June) 1918.

149. Cherry, T. H., and E. C. Langrock: Relation of Hemolysis in the Transfusion of Babies with Mothers as Donors, J. A. M. A. **66**:626 (Feb. 26) 1916.

150. Helmholz, H. F.: Intravenous Aspirations and Injections Including Transfusions; Longitudinal Sinus as Place of Preference in Infancy, Am. J. Dis. Child. **10**:94 (Sept.) 1915.

151. Goldbloom, Alton: New Apparatus for the Puncture of the Superior Longitudinal Sinus, Am. J. Dis. Child. **16**:388 (Dec.) 1918.

152. Zingher, Abraham: Transfusion of Citrated Blood in Children, Am. J. Obst. **71**:1015 (June) 1915.

In treatment of hemorrhagic disease of the new-born, Howard[153] recommends a simple technic of transfusion. The blood is withdrawn from the vein of the donor by the use of a twenty c.c. sterile ground glass syringe containing 2 c.c. of a 10 per cent. sodium citrate solution. The needle is then inserted into the superior longitudinal sinus of the infant at an angle of twenty-five degrees with the scalp, and emptied slowly. The operation may be repeated several times if necessary. The simplicity of the apparatus and technic greatly recommends this method. The author reports a case treated in this manner.

Berghausen[154] reports a case of hemorrhage in a new-born in which he introduced between 150 c.c. and 200 c.c. of the father's blood in 2 per cent. citrate solution into the superior longitudinal sinus of the infant. The patient showed marked immediate improvement. He believes the method is so simple and efficient that it will probably be the one of choice in the future.

Knox[155] reports a case of a two-days old infant who vomited a considerable amount of bloody fluid and passed tarry blood-stained material, evidently from hemorrhage in the intestinal tract. Fifty c.c. of mother's blood was received into 60 c.c. of sodium citrate solution, 15 c.c. of this being introduced into the superior longitudinal sinus of the child, and the balance in small quantities intramuscularly into the buttocks. On the second day following, the stools were yellow and the child made a satisfactory recovery.

Jarvis[156] reports thirteen cases of hemorrhagic disease. Among these cases were seven patients ranging in age from three days to two months. There were various hemorrhages from the bowels, nose, mouth, ears, naval, and in some cases purpuric spots on the skin. He treated all patients with injections of whole blood, his method being to draw about 20 c.c. of blood from the father or some male relative and inject it into the child's buttock, repeating as often as necessary. Two of these children grew steadily worse and died in spite of repeated injections. In all others the bleeding was stopped.

A rather unique scheme of giving blood in cases of hemorrhage of the new-born is suggested by Hubbard.[157] Calling attention to the

153. Howard, W. S.: Hemorrhage of the New-born; A Simple Method of Transfusion in, with Report of a Case, J. A. M. A. **65**:1365 (Oct. 16) 1915.

154. Berghausen, Oscar: Hemorrhagic Disease in New-born, J. A. M. A. **70**:514 (Feb. 23) 1918.

155. Knox, J. H. Mason: A Case of Hemorrhagic Disease in the New-born Treated by Indirect Transfusion, Arch. Pediat. **34**:771 (Oct.) 1917.

156. Jarvis, H. Gildersleeve: Treatment of Hemorrhagic Disease, Boston M. & S. J. **171**:165 (July 23) 1914.

157. Hubbard, J. C.: Hemorrhagic Disease of New-born, Suggestion in Treatment of, Boston M. & S. J. **172**:865 (June 10) 1915.

rapid absorption of fluid introduced into the abdominal cavity, he thinks it might be practical to inject blood in this manner rather than to try to transfuse or use subcutaneous injections. The author evidently has not found this method successful, as there are no further reports.

Before the treatment by whole or citrated blood had become well established, these cases of hemorrhage were largely treated by serum injections. Manning[158] reports six cases of hemorrhage in the newborn in which he used the serum treatment. Two of the six patients succumbed, one being of intracranial type and the other being a streptococcic septicemia. He used subcutaneous injections of whole blood in only one case, repeated injections of horse serum being employed in the other cases. There were no reports of the Wassermann reaction on the children.

Four cases of hemorrhagic disease in the new-born treated by horse serum are reported by Hymanson.[159] He points out that the transfusion method may sometimes be impractical, as a mother is often too weak to act as a donor. The father is often away or unwilling, and a healthy stranger is hard to obtain at the moment. The author concludes that the coagulation time of blood is delayed in this disease and that fresh horse or rabbit serum will serve the purpose for injections almost as well as human serum. In the new-born with hemorrhagic disease horse serum should be administered early and repeatedly until the bleeding ceases. The injurious effect caused by foreign serum has been greatly exaggerated.

Triollet[160] states that he has found remarkably effective in the treatment of gastro-intestinal hemorrhage the combined use of calcium chlorid, one teaspoonful of 2 per cent. solution, every two hours, by mouth, and a subcutaneous injection each day in the scapular region of 20 c.c. of gelatinized serum. No food is allowed, but a few teaspoonfuls of boiled water are given every two hours. The child is kept warm and treatment is continued for a day or two after hemorrhage ceases. Then the breast is given cautiously. The gelatinized serum is sterilized at 120 C. for thirty minutes. If there is a suspicion of syphilis, specific treatment is also given.

Brindeau[161] advocates the free use of the trephine in cases of cere-

158. Manning, J. B.: Six Cases of Hemorrhage in Newly Born Babies, Arch. Pediat. **32**:838 (Nov.) 1915.

159. Hymanson, A.: Four Cases of Hemorrhagic Disease in the Newly Born Treated by Horse Serum, Am. J. Obst. **71**:698 (June) 1915.

160. Triollet, L.: Treatment of Gastro-Intestinal Hemorrhage in the Newborn, Arch. mens. d'obst. et de gynéc., Paris **5**:354, 1916.

161. Brindeau: Trephining in the Newly Born, Arch. mens. d'obst. et de gynéc. **7**:103 (April-June) 1918.

bral hemorrhage. He believes that the intervention by this method is much less formidable than has been believed, and is indicated in all cases of depression or meningeal hemorrhage. He reports four cases, two of which were lifesaving.

BIRTH INJURIES

According to Hannah,[162] the day of birth records the highest death rate of all days. Schlutze has established that 5 per cent. of all children are stillborn or die during labor, and 1 per cent. die soon after birth as a result of trauma of labor. DeLee states that in the United States 75,000 children die annually during delivery, and that a large number of these die from asphyxia and injuries during birth. The others die from syphilis, alcoholism and congenital defects. Porter says that 17 per cent. of the inmates of feebleminded institutes of Indiana give a history of difficult births, and 4 per cent. a history of forceps operation.

It is in contracted pelves that the child suffers most, and it is here that by use of forceps we sometimes get depressed fractures, concussion of the brain, or intracranial hemorrhage, which may in later life cause paralysis, epilepsy, etc. Often asphyxia neonatorum is due to a long and difficult labor, which is more or elss productive of trauma. Any condition during labor which will cut off the supply of oxygen or compresses the brain unduly will produce asphyxia. Any condition that will lessen the placental area, that will compress the placenta, as in case of after-coming head, that will cause the separation of the placenta in part or completely, or that will cause pressure on the umbilical cord, will cause hypercarbonization. Internal or extreme pressure of the brain, whether due to forceps, hematoma, or depressed fracture, may prevent return flow of the blood from the fetus to the placenta, resulting in hypercarbonization. It is a bad omen when lumpy or sea green colored meconium is found in the liquor amnii of a cephalic presentation. It is significant of intra-uterine asphyxia. Among other conclusions Dr. Hannan says that an internal hematoma causing internal pressure should be removed early, or permanent pathologic destruction of the nervous system will take place, causing imbecility, epilepsy and deformity.

In an article on the influence of labor on the brain development of the child, Stein[163] says that protracted labor is responsible for brain injury, which results in defective mentality, and idiocy, as well as

162. Hannah, C. R.: Injuries to the Infant Produced at Birth, Texas State J. M. **11**:539 (Feb.) 1916.

163. Stein, Arthur: The Influence of Labor on the Brain Development of the Child, J. A. M. A. **69**:334 (Aug. 4) 1917.

epilepsy, and cerebral palsies. The injury is more likely to occur when the children are born asphyxiated. Children with faulty heredity are more likely to show mental defects after prolonged than after ordinary labor. Forceps correctly applied are a beneficent weapon against the abnormal prolonged passage of the child's head through the pelvic canal. Pituitary solution in small doses is of great value.

In an excellent review of the literature on what is known as Little's disease, Wall[164] believes that this title should represent cases of congenital spastic rigidity or paralysis, having their origin before or during the act of birth, because of causes operating within the central nervous system, more especially within the cerebrum. Prematurity, syphilis, hemorrhage due to birth trauma, and asphyxia neonatorum are given as prominent factors, causing the disease. He reports a number of cases illustrating the symptoms. The prognosis, on the whole, is not very hopeful, depending entirely on the amount of involvement. Treatment has been mostly orthopedic. He calls attention, however, to the recent work of Sharpe and Farrell, which may offer relief in especially selected cases.

Sharpe[165] operated on children that had gone through difficult labor and that revealed changes in the optic disk and in the spinal fluid indicative of increased intracranial pressure. The most satisfactory results were obtained in those cases having no impairment of mentality. Hemorrhage of the new-born resulting from rupture of a tributary vein of the superior longitudinal sinus from the application of forceps to the child's head, or from other rough handling, is usually cortical or subcortical. Intracranial hemorrhage accounts for 70 per cent. of the cases of spastic paralysis. Trephine openings are made and the dura divided and left open. He performed subtemporal decompression on one or both sides, and stated that cases with increased intracranial pressure offered hope of improvement, from 8 to 12 cm. being considered normal measures of pressure. In another paper Sharp[166] reports the operative treatment of 219 selected cases. These were selected from a group of 954 examined. These cases of spastic paralysis showed increased intracranial pressure by an ophthalmoscopic examination and by measurement of pressure of cerebrospinal fluid by lumbar puncture. Mentally defective, microcephalic children, or those affected with so-called Little's disease, which form

164. Wall, Joseph S.: Little's Disease (Congenital Rigidity of Infancy), Arch. Pediat. **33**:812 (Nov.) 1916.

165. Sharpe, Wm.: Results of Cranial Decompression in Selected Types of Cerebral Spastic Paralysis Due to Hemorrhage, Med. Rec. **89:** (June) 1916.

166. Sharpe, Wm.: Results of Cranial Decompression in Selected Types of Cerebral Spastic Paralysis Due to Hemorrhage, New York State J. M. **16**:475 (Oct.) 1916.

one-half of all cases of spastic paralysis, were not subjected to operation. Operation was performed in those cases which presented a history of difficult labor and showed definite signs of intracranial pressure, as indicated by changes in the fundus of the eye and confirmed by increased pressure of the spinal fluid. He again calls attention to the frequency of intracranial hemorrhage of the new-born. This is frequently of venous origin, especially from the tributaries of the superior longitudinal sinus, or the sinus itself may be ruptured by extreme overlapping of the parietal bones during parturition. If the hemorrhage occurs on, rather than within, the cortex, the cortex is not damaged, and its nerve cells will be able to functionate normally if the pressure of the overlying hemorrhagic clot is removed. In the majority of cases the hemorrhage extends over one hemisphere alone, the spastic paralysis involving one leg or one arm opposite the affected hemisphere. Absorption of the clot may have taken place to an extent where only a few fibrous strands remain; often these patients develop epilepsy.

Matzinger,[167] in his article on the operative benefit for cerebral defects, states that proof was wanting that meningeal hemorrhage gave rise to diplegia. He considers hemorrhage a definite indication for operation, and thinks the eye grounds give important operative indications in cases of infantile cerebral paralysis.

Findings in a study of 470 cases of obstetric paralysis are reported by Sever.[168] Antedating this paper, fifty-eight authors had reported a total of 457 cases. Sever's article is a careful review of the literature from the first report by Smellie in 1768 to the present, and an excellent discussion of the various theories brought forth during this period. Among the earlier theories which had considerable following, was that the injury to the brachial plexus was produced by forceps pressure. Another theory was that the injury was caused by pressure of the fingers in the axilla, another that traction on the arm in breach delivery was responsible, yet another was pressure on Erb's point, especially on the energetic application of the so-called Prague's grip. Seeligmuller thought that pressure from hemorrhage around the plexus often produced paralysis. Kustner believed that the trouble was uniformly due to fracture of bones or separation of the epiphysis. Danchez thought the condition was spontaneous from pressure on the circumflex nerve. Destrox thought that the condition was a pseudo-paralysis and involved no injury to the nerve, but that the arm was

167. Matzinger, Herman G.: Types of Cerebral Defects in Children that may be Benefited by Operation, New York State J. M. **16**:484 (Oct.) 1916.

168. Sever, James Warren: Obstetric Paralysis, Am. J. Dis. Child. **12**:541 (Dec.) 1916.

held motionless because of the bruising, and consequent pain, or the result of a bone injury. Fieux was the first to offer proof that the condition was due to the separation of the upper roots of the brachial plexus, probably caused by a sharp pulling of the head to the right or the left while delivering the shoulders. Fibers of the suprascapular nerve always ruptured first. Schoemaker conducted experiments on cadavers which verified the work of Fieux. He thought also that pressure could be exerted on the plexus by squeezing it between the clavicle and first rib and the spine. Thomas reports two cases of bilateral paralysis of the lower arm type following difficult labor with face presentation, in which he believed the injury to be the result of excessive lordosis or hyperextension. Bullard suggests that asphyxia is a favorable condition for the production of the paralysis. Lange offered the theory that the paralysis is due to the tearing of the capsule of the shoulder joint. This theory was elaborated by T. T. Thomas and others. However, Sever seems to prove conclusively that the injury is due primarily to rupture of the nerve sheath and the fraying out of the nerve fibers. The healing nerve sheath forms a cicatrix, which strangulates the nerve fibers, and prevents their regeneration. Nerve repair of the brachial plexus has been attempted many times with no favorable results reported. The author has, however, accomplished a great deal by division of the tendons of the contracted muscles involved. In conclusion, he says that obstetric paralysis is due to a stretching or tearing of the fifth and sixth cervical roots of the brachial plexus. It occurs in boys as frequently as in girls. It occurs more often on the right than on the left side. The upper arm type is much more frequent than the lower arm type. It affects both arms very infrequently. It is practically always associated with difficult labor, in which ether and forceps have been used, and force has been applied. Not uncommonly is the baby asphyxiated. Head presentation shows the greater percentage of occurrences of both types of cases. It may rarely be associated with fracture of the clavicle, but is not the result of a fractured humerus or a dislocated shoulder joint. The prognosis for a useful arm is good in the upper arm type and bad in the lower arm type.

Thomas[169] divides the treatment of cases of obstetric paralysis into the operative type and nonoperative type. Two operative procedures have been employed, the operation on joints to relieve limitation of motion from ligamentous contracture, and the transplantation of strong muscles to supplement the action of weak ones. The best results are from the transplanting of the pectoral or the trapezius

169. Thomas, J. J.: Obstetrical Paralysis with Especial Reference to Treatment, Boston M. & S. J. **170**:513 (April 2) 1914.

into the deltoid, and the excision of a portion of the brachial plexus at Erb's point and the suture of the nerve trunks, as first practiced by Kennedy and later by Taylor. This method should not be used earlier than 2½ or 3 years of age, and not later than 7 or 8 years of age. It should be reserved for severe cases. The nonoperative method of treatment consists of massage and educational gymnastics. Before the age of two months very gentle massage and irregular movements, such as moving the baby's hand and arm in various directions in imitation of voluntary movements, are employed.

Rogers[170] suggests an osteotomy of the upper portion of the humerus about two inches below the shoulder joint followed by one-fourth rotation of the whole arm below the line of fracture.

Smith[171] reports two cases of obstetric paralysis involving only the musculospiral nerve on one side. In neither case was there any history of syphilis. In one case the labor was precipitate and in the other a hard, long drawn out operation. In neither case was there any other deformity or injury. Both cases were observed shortly after birth, and the babies made a spontaneous recovery. In one case there was a small tumor at the seat of injury to the nerve. Smith discusses the various theories of birth paralysis and concludes that the only tenable theory in his cases is that of injury during birth.

In Gordon's[172] case of birth paralysis the lesion was bilateral and confined to the flexors of the wrist exclusively.

Truesdell[173] reports the study of twenty-four birth fractures of the humerus with roentgenographic and clinical examinations at intervals until the final outcome. In some cases the deformity was extreme at the onset, but in no case did the roentgenogram fail to show a progressive elimination of the deformity, recording the transition effected by nature, from a distorted bone to one finally indistinguishable from its fellow of the opposite side. The prevailing deformity was an external angulation attributed to the action of the deltoid. The line of fracture was practically transverse, at or near the center of the shaft of the bone. Various devices were used in treatment, but a broad band of adhesive plaster passed around the chest fixing the injured arm to the side of the body with forearm flexed and hand brought toward the opposite shoulder proved the most

170. Rogers, M. H.: Operation for the Correction of the Deformity Due to Obstetrical Paralysis, Boston M. & S. J. **174**:163 (Feb. 3) 1916.

171. Smith, L. Howard: Paralysis, Two Cases of Obstetrical Involving Only the Musculospiral Nerve, Am. J. Dis. Child. **11**:133 (May) 1916.

172. Gordon, Alfred: Birth Palsy, An Unusual Form of, J. A. M. A. **63**: 2282 (Dec. 26) 1914.

173. Truesdell, E. D.: Fractures of Humerus, Birth, from Wards of New York Lying-in Hospital, Arch. Pediat. **32**:669 (Sept.) 1915.

satisfactory. He concludes that in these cases a temporary paralysis of the musculospiral nerve is usual and displacement of the fragments always occurs. Every effort should be made to combat deformity, but nature will so supplement the treatment as to insure satisfactory results, eliminating all deformity by the end of the second year. He reproduces a score of excellent prints. In a second paper, Truesdell remarks that where fracture is complicated by Erb's paralysis less improvement is noted. Ten cases of fracture of the clavicle with slight displacement are reported in which union took place promptly; also two cases of fracture of the femur. In the latter cases the functional demands are so much greater than in the humerus fractures that close observation with traction are necessary to prevent permanent deformity.

Russell[174] describes a suspension apparatus for the proper reduction of the fracture of the femur in new-borns. To an overhead frame is attached a pulley suspension apparatus so balanced by weights that the child may be moved about in bed without disturbing the position and traction of the injured leg.

HYDROCEPHALUS

Frazier[175] classifies hydrocephalus as obstructive, nonabsorptive, hypersecretive and occultus. The author has a definite technic for the differentiation of the nonabsorptive and obstructive types based on the appearance of phenolsulphonephthalein in the urine after injection into the ventricles or lumbar subarachnoid. The technic is described in detail with a case report attached. The use of the thyroid extract in the hypersecretive form may give good results, and in the obstructive form puncture of the corpus callosum is advocated. He proposes the practicability of providing permanent drainage into the plural cavity for cases of the nonabsorptive type.

Browning,[176] in discussing the cause of hydrocephalus, points out that for a time after birth the child has but a very limited efferent system for the spinal fluid. This is the favorite time for the ocurrence of the disease. The explanation for this is largely in the closure of the spinal outlets from the time of birth and the imperfect development of the pacchionian bodies at this period.

---

174. Russell, Thos., and Harry S. Reynolds: Apparatus in Birth Fracture of Femur, J. A. M. A. **68**:1902 (June) 1917.

175. Frazier, C. H.: Types of Hydrocephalus; Their Differentiation and Treatment, Am. J. Dis. Child. **11**:95 (Feb.) 1916.

176. Browning, Wm.: Anatomical Cause of the Frequency of Hydrocephalus in Childhood, Med. Rec. **89**:959 (May 27) 1916.

Oden[177] discusses the possibility of delivery through a contracted pelvis as the cause of hydrocephalus developing after birth. There may be a mechanical occlusion of the foramen of Magendie or the aqueduct of Sylvius from passage through the contracted pelvis. A case is reported which would seem to bear out this conclusion. Two children delivered through the contracted pelvis of the mother developed postpartum hydrocephalus, while a third child delivered through a cesarean section, developed no abnormal symptoms. He believes that this should be the method of choice in later pregnancies when there is a contracted pelvis and hydrocephalus has developed after a previous difficult labor.

SPINA BIFIDA

According to Gale,[178] spina bifida occurs about once in every thousand births; 23 per cent. are sacral, 24 per cent. lumbar, 29 per cent. lumbosacral, 4½ per cent. dorsal and 9½ per cent. cervical. Prognosis is most favorable in the meningocele type, and next in the myelocystocele type. Many patients who survive radical treatment die of hydrocephalus, or of the secondary effects of an existing paralysis. In the cases of those patients who are at least 5 years old, operation may be done fairly safely, because few patients who cannot be operated on reach the age of five years. Mayo-Robson reports a fatal result in over 35 per cent. of cases operated within the first few months of life, while in children over five years, the mortality is only 4.7 per cent. Local anesthesia should be used, and bone or other foreign bodies to fill the cleft are not recommended.

Iruegas[179] reports an operation eight hours after birth without anesthetic. The spina bifida was of the meningomyelocele type. All nerve elements should be pushed back into their normal place when possible. To close the aperture he sutures together the inner lips of the sack around the orifice of communication, then sutures the external lips above this, then sutures the meninges at the point where they were opened on a level with the skin. Wound must be protected against soiling with some impermeable covering.

Cate's[180] article reports the result of operation on nine additional cases of spina bifida and sums up the results, with seven cases previously reported. The youngest patient was 21 days old, and the oldest

---

177. Oden, Rudolph: The Possible Relation of a Contracted Pelvis to Hydrocephalus Developing After Birth, J. A. M. A. **64**:816 (March 6) 1915.

178. Gale, S. S.: Spina Bifida, Report of Cases, West Virginia M. J. **9**:145 (Nov.) 1915.

179. Iruegas, D. F.: Operative Treatment of Meningomyelocele, Arch. Espan. de Pediat. **2**:350 (June) 1918.

180. Cates, B. B.: Spina Bifida, Boston M. & S. J. **174**:420 (March 23) 1916.

patient was 34 years old. He does not believe that the age of the patient is as important a factor in determining the personal equation as is the physical condition. Of the sixteen patients, only ten survived beyond a postoperative period of three months. Of these ten, one died four months and one eight months after operation of pneumonia, and one died six months after operation from hydrocephalus.

Harmer[181] gives the detailed operative procedure for spina bifida of the different types, and sums up the results of his experience in thirty-four cases. Sixteen patients died, a mortality of 47 per cent. Of the patients who lived four exhibited some degree of paralysis and three an increasing hydrocephalus. The earlier the patients are operated on the better. It should be done within the first few hours after birth, if possible.

Two successfully operated cases of the meningocele type, one patient 11 months of age and the other 14 months of age, are reported by Norton.[182] He believes that operation is indicated in all cases, as 90 per cent. of children thus afflicted die within the first year of life if untreated. The main points in the operation are careful separation of nerve roots from sac wall and base, perfect hemostasis and asepsis, and absolute closure of the sac so that leakage of spinal fluid cannot occur. Celluloid plates, bone grafts or silver plates to close the bony defect add greatly to the operation, and are not necessary, as the muscles and fascia of the back afford a strong and efficient covering. Prognosis is good in operating even though the closing is followed by hydrocephalus, as this usually subsides.

Beer[183] reports a case of a boy, 11 days old, who had urine rete tion due to paralysis associated with a well marked spinal cord lesion.

TUBERCULOSIS

An interesting case with necropsy findings of miliary tuberculosis in a new-born child which died on the eleventh day of life is reported by Grulee and Hames.[184] Clinical symptoms were increased temperature, convulsions, vomiting, loss of weight and enlarged liver and spleen. At necropsy there was found miliary tuberculosis of all organs and the mesenteric lymph nodes. New-born tuberculosis is divided into two forms, namely, pulmonary and abdominal. Both forms are

181. Harmer, T. W.: Spina Bifida and Allied Malformations, Boston M. & S. J. **177**:353 (Sept. 17) 1917.

182. Norton, W. A.: Report on Two Successfully Operated Cases of Meningocele, Am. J. Obst. **77**:169 (Jan.) 1918.

183. Beer, E.: Chronic Retention of Urine in Children, J. A. M. A. **65**:1709 (Nov. 13) 1915.

184. Grulee, C. G.: Harms, F., Tuberculosis as a Disease of New-borns, Am. J. Dis. Child. **9**:322 (April) 1915.

of the miliary type. No authentic cases of localized tuberculosis in the new-born have been reported. Ordinary tests are of little value in diagnosis. The von Pirquet test has proved negative almost without exception. Blood cultures or blood injected into animals have been suggested as a means of diagnosis, but the condition is rarely suspected and the patient would die before the animal which was injected. The prognosis in tuberculosis of the new-born has been, up to this time, always fatal.

Ugon[185] reports two cases of tuberculosis evidently contracted by the infants from the tuberculous mothers. Both were a little prematurely born, and were suckled by their mothers. One developed signs of pulmonary tuberculosis one month after birth, the other in the third month. Tuberculin tests were positive on the fiftieth day in one case and on the ninetieth day in the other, previous tests having been negative. The clinical diagnosis of pulmonary tuberculosis was confirmed at necropsy.

In their review of 103 cases, Norris and Landis[186] find that the child of the tuberculous mother is not usually an hereditary weakling. The high mortality among these infants is due to the fact that they are usually artificially fed and do not have proper mother's care. Infants should not be nursed by tuberculous mothers, and should be especially guarded against infection.

McSweeny,[187] reporting on the tuberculosis ward for pregnant mothers at Sea View, says that the child should be removed immediately after birth to a distant pavilion, should not be permitted to nurse and should be fed artificially from birth.

In Harbitz'[188] case the mother died of tuberculosis twenty-eight days after child's birth. There was tuberculosis of the endometrium at the placental site so that transmission of the tubercle bacilli directly into the blood stream could easily have occurred. The uterus probably had become infected from old tuberculous lesions found in the tubes. The child was removed from the mother immediately after birth, but died on the twenty-fifth day. Extensive tubercular changes were found in the lungs and lymph nodes. From his experience, Harbitz believes that an infant dies very shortly after contracting a tubercular infection. If a child up to three months of age has lived even for a

185. Ugon, M. A.: Tuberculosis in the New-born, Arch. Latino-Amer. de Pediat. **12**:213, 1918.

186. Norris, C. C., and Landis, H. R. M.: Pregnancy and Pulmonary Tuberculosis, J. A. M. A. **70**:362 (Feb. 9) 1918.

187. McSweeny, E. S., and Wang, S. L.: Childbirth and Tuberculosis, J. A. M. A. **70**:368 (Feb. 9) 1918.

188. Harbitz, F.: Congenital Tuberculosis, München med. Wchnschr. **60**: 741, 1913.

short time in a tuberculous environment, no conclusion can be drawn as to whether the infection occurred before or after birth. On the other hand, infants known to have congenital tuberculosis have lived to be one year old and even more. Necropsy of six infants less than five weeks old, all born of tuberculous mothers, produced no evidence of tuberculosis. Infection before birth is considered very rare.

One of the earliest positive von Pirquet reactions is reported by Zarfl[189] in a 17-day-old child. The mother of the child was tuberculous. The child's weight decreased rapidly. The liver, spleen and inguinal lymph nodes became much enlarged. The lung findings were negative. Zarfl believes this is the earliest positive von Pirquet reported, and thinks that its early appearance is proof that the disease was congenital, because at least four weeks must elapse after an infestion before a positive tuberculosis reaction can be obtained.

Von Weller[190] reports two cases of miliary tuberculosis of the placenta in which apparently healthy children were born. Neither mother had other active signs of the disease. One of the children remained under observation for eighteen months with no apparent evidence of the disease.

A case of tuberculosis following ritual circumcision is reported by Reuben.[191] The child, then nine weeks old, had been circumcised by a Mohel when eight days old. Five weeks later a swelling appeared in the inguinal glands of the right groin. Four small tubercular masses appeared on the circumcision scar, and the von Pirquet reaction was positive. The sputum of the Mohel was examined and found to be loaded with tubercle bacilli. In a review of forty-two cases it was found that the wound usually ulcerates within from seven to fourteen days after operation. Inguinal glands enlarge in from two and one half to eight weeks after operation, usually breaking down. The majority of infants die before the end of the third year from tuberculous meningitis or miliary tuberculosis. Those who recover invariably present manifestations of tuberculosis in the later life. Usually this is tuberculosis of the bones or lymph glands. Early excision of the tuberculous tissues of the penis and of the inguinal glands on both sides may give good results.

---

189. Zarfl, M.: Congenital Tuberculosis, Jahrb. f. Kinderh. **77**:95, 1913.

190. Von Weller, C.: Two Additional Cases of Miliary Tuberculosis of the Placenta, with Clinically Latent Tuberculosis of the Mother, Arch. Int. Med. **17**:509 (April) 1916.

191. Ruben, M. S.: Tuberculosis from Ritual Circumcision, Arch. Pediat. **34**:186 (March) 1917.

CURRENT PEDIATRIC LITERATURE

ANATOMY AND PHYSIOLOGY

Mammary Gland, Studies of. IV. Histology of Mammary Gland in Male and Female Albino Rats from Birth to Ten Weeks of Age. J. A. Myers.
Am. J. Anat. **25**:395 (July 15) 1919.

Ribs, Cervical; Two Cases. C. Pelfort.
Arch. espan. d. Pediat. **3**:154 (March) 1919.

Scrotum: Two Testes and Uterus in Right Portion of Scrotum, Congenital Malformation in. D. W. MacKenzie.
Canad. M. A. J. **9**:641 (July) 1919.

METABOLISM AND NUTRITION

Energy Requirements of Children from Birth to Puberty. F. G. Benedict.
Boston M & S. J. **181**:107 (July 31) 1919.

Fat Metabolism of Infants and Young Children. III. Fat in Stools of Children on a Mixed Diet. L. E. Holt, A. M. Courtney and H. L. Fales.
Am. J. Dis. Child. **18**:107 (Aug.) 1919 .

Feeding, Infant. L. S. Dietrich.
Iowa State M. J. **9**:208 (July 15) 1919.

Milk, Model, for Young Children. Rocaz.
J. de med. de Bordeaux **90**:249 (June 25) 1919.

Milk, Women's; Study of Lactose, Fat and Protein Content of. W. Denis and F. B. Talbot.
Am. J. Dis. Child. **18**:93 (Aug.) 1919.

Orange Juice, Constipating Qualities of. H. J. Gerstenberger and W. M. Champion.
Am. J. Dis. Child. **18**:88 (Aug.) 1919.

Rachitis and Digestive Disturbance. P. Pereda y Elardi.
Med. Ibera **7**:161 (May 31) 1919.

HYGIENE AND SANITATION

Hygiene, Child; Development of Bureau of, by State Board of Health of South Carolina. R. A. Dodd.
J. S. Carolina M. A. **15**:496 (July) 1919.

Health, Public and Child Hygiene. E. S. Hamblen.
J. M. S. New Jersey **16**:118 (April) 1919.

DISEASES OF THE NEW-BORN

Erysipelas in Newly Born. Lesne.
Progres med. **34**:192 (May 10) 1919.

Hemorrhage of New-Born, Cerebral. M. Warwick.
Am. J. M. Sc. **158**:95 (July) 1919.

New-Born, Review of Recent Literature on. N. O. Pearce.
Am. J. Dis. Child. **18**:133 (Aug.) 1919.

ACUTE INFECTIOUS DISEASES

Diphtheria, the Uncontrolled. B. W. Carey.
Boston M. & S. J. **181**:92 (July 24) 1919.

Influenza in Infants. C. A. Achard.
Bull. de la soc. med. d. hôp. **43**:455 (May 16) 1919.

Malaria in Children. R. Gomez Ferrer.
Arch. espan. d. Pediat. **3**:129 (March) 1919.
Measles, Epidemic of Relapsing. G. Desbouis.
Bull. de la Soc. med. d. hôp. **43**:468 (May 16) 1919.
Rheumatism, Outcome after Tonsillectomy for. H. Nordlund.
Hygiea **81**:497 (June 16) 1919.
Scurvy in Infants, Initial Phase of. O. Gozzolino.
Pediatria **27**:321 (June) 1919.
Spasmophilia, Cerebral Fluid with. G. Genoese.
Riforma med. **35**:477 (June 14) 1919.
Spasmophilia in Children. T. Luzzatti.
Policlinico **26**:769 (June 22) 1919.
Whooping Cough, Hemorrhagic Meningeal Complications with. A. F. Canelli.
Pediatria **27**:351 (June) 1919.

TUBERCULOSIS AND SYPHILIS

Eruption, Papulous, of Probably Tuberculous Origin. P. M. Barlaro.
Prensa med. Argentina **5**:326 (April 30) 1919.
Syphilis, Asylums for Children with Inherited. F. Balze.
Bull. de l'Acad. de med., Par. **81**:811 (June 17) 1919.
Syphilis, Inherited; Athrepsia in. Ribadeau-Dumas and Faroy.
Le Nourrisson **7**:146 (May) 1919.
Syphilis, Inherited; Latent Meningeal Reactions in. Lavergne.
Le Nourrisson **7**:172 (May) 1919.
Syphilis, Tardy Inherited. R. E. Parody.
Prensa med. Argentina **5**:325 (April 30) 1919.
Tuberculosis in Children, Stages of. J. P. Garrahan.
Prensa med. Argentina **5**:321 (April 30) 1919.
Wassermann Reaction with Human Milk. C. L. Rusca.
Pediatria **27**:343 (June) 1919.

RESPIRATORY SYSTEM

Empyema in Children, with Special Reference to Diagnosis. F. S. Churchill.
Boston M. & S. J. **181**:87 (July 24) 1919.
Glandular Disease, Tracheobronchial. H. Mery and Others.
Bull. la Soc. med. d. hôp. **43**:471 (May 16) 1919 .
Hay-Fever in Children. W. Scheppegrell.
Med. Rec. **96**:97 (July 19) 1919.

GASTRO-INTESTINAL SYSTEM

Esophagus in Child, Stricture of. E. M. Eberts.
Canad. M. A. J. **9**:630 (July) 1919.
Gastric Secretion in Young Children. A. T. B. Jacobsen .
Ugesk. f. Læger **81**:863 (May 22) 1919.
Pancreas in Children, Insufficiency of. C. S. de los Terreros.
Siglo med. **66**:353 (May 3) 1919.
Pin in Child's Appendix. R. A. Rivarola and A. Sein.
Semana méd. **26**:506 (May 15) 1919.
Pylorus, Congenital Stenosis of. A. B. Marfan.
Le Nourrisson **7**:129 (May) 1919.
Safety Pin, Open; Swallowed by an Eight Months Old Child and Passed Through the Rectum after Four Days. S. Weiss.
Med. Rec. **96**:189 (Aug. 2) 1919.
Teeth, Abscessed in Children; Bacterial Findings in. A. L. Smith and R. W. Ludwick.
Nebraska M. J. **4**:131 (May) 1919.
Vomiting of Infants. G. Schreiber.
Paris med. **9**:430 (May 31) 1919.

BLOOD AND CIRCULATORY SYSTEM

Gallop Rhythm in Children. L. Morquio.
Ann. de la Facul. de med. **4**:168 (March-April) 1919.

Stenosis of Pulmonary Artery, Congenital. O. Josue.
Bull. soc. med d. hôp. **43**:408 (May 9) 1919.

NERVOUS SYSTEM

Brain, Excitability of, in Young Infants. G. Bergmark.
Upsala Läkaref. Förh. **24**:420 (June 17) 1919.

Brain (Telangiectasis) and Associated Cerebral Movements, Congenital Tumor of. W. G. Spiller.
Arch. Neurol. & Psychiat. **2**:50 (July 1) 1919.

Delinquency, Mental Disease and. Report of Special Committee of New York State Comission of Prisons. V. V. Anderson.
Ment. Hygiene **3**:177 (April) 1919.

Encephalitis, Histopathology of Epidemic ("Lethargic"). I. J. Sands.
Arch. Neurol. & Psychiat. **2**:24 (July 1) 1919.

Encephalitis, Postinfluenzal. H. Heiman.
Am. J. Dis. Child. **18**:83 (Aug.) 1919.

Epilepsy, Relationship of Convulsions in Infancy and Childhood to.
Am. J. Dis. Child. **18**:73 (Aug.) 1919.

Epilepsy, Rôle of Pituitary Gland in. B. R. Tucker.
Arch. Neurol. & Psychiat. **2**:192 (Aug. 1) 1919.

Meningitis, Cerebrospinal; Cause of a "Sporadic" Case of. H. L. K. Shaw.
Am. J. Dis. Child. **18**:101 (Aug.) 1919.

GENITO-URINARY SYSTEM

Genital Organs, Disease of, in Little Girls. J. Blanc Fortacin.
Arch. espan de Pediat. **3**:146 (March) 1919.

Nephritis in Children, Retention of Chlorids and Urea in. P. Nobecourt.
Presse med. **27**:341 (June 23) 1919.

Nephritis, Influenzal, in Children. G. Giacobini.
Semana med. **26**:406 (April 17) 1919.

BONES AND JOINTS

Arthritis, Classical Case of Hemophilic. M. G. Wilson.
Am. J. Dis. Child. **18**:127 (Aug.) 1919.

Spine in Growing Children, Curvatures of. E. H. Bradford.
New York M. J. **60**:1 (July 5) 1919.

EYE, EAR, NOSE AND THROAT

Blind, Education of the Young. E. M. Van Cleve.
New York M. J. **60**:54 (July 12) 1919.

Otitis Media, Acute. J. Giannetto and P. J. Martino.
An. Facultad. de med. **4**:194 (March-April) 1919.

Tonsillectomy, Apothesine Anesthesia in. J. Coleman.
New York M. J. **60**:19 (July 5) 1919.

Tonsillectomy for Rheumatism, Outcome after. H. Nordlund.
Hygiea **81**:497 (June 16) 1919.

Tonsils, Especially in Children, Unwarranted Sacrifice. H. M. Harrison.
Illinois M. J. **36**:14 (July) 1919.

Tonsillitis in Children, Significance of. C. P. Gray.
New Orleans M. & S. J. **72**:16 (July) 1919.

Tonsils in Young Children, Suggested Alternative to Operation for Adenoids and Enlarged. J. Donelan.
J. Laryngol., Rhinol. & Otol. **34**:229 (July) 1919.

MISCELLANEOUS

Asylum for Infants. Rocaz.
J. d. med. de Bordeaux **90**:246 (June 25) 1919.

Backward Child, Helping the. F. W. Barrows.
New York State J. M. **19**:284 (July) 1919.

Boarding Out Infants in the Country. P. F. Armand-Delille.
Le Nourrisson **7**:166 (May) 1919.

Child, How to Avoid Spoiling the. L. F. Barker.
Ment. Hygiene **3**:240 (April) 1919.

Children's Pavilion for Contagious Disease. E. Feer.
Cor.-Bl. f. schweiz. Aerzte **49**:777 (May 24) 1919.

Fever, Postoperative in Infants. P. de Elizalde.
Semana med. **26**:404 (April 17) 1919.

Mongolism in One of Twins, Case of. H. Swanberg and H. A. Haynes.
Arch. Neurol & Psychiat. **1**:717 (June 1) 1919.

Mortality, Infant; at Bordeaux. Chambrelent.
J. de med. de Bordeaux **90**:240 (June 25) 1919.

Mortality, Infant; Countries That Have High Regions Should Utilize Them to Reduce. J. Santos Fernandez.
Cron. méd. chirurgica **45**:117 (April) 1919.

Sarcomatosis, Case of Congenital Multiple. J. A. Perera.
Lancet **2**:14 (July 5) 1919.

Temperature Variations in Infancy and Early Childhood. I. A. Abt.
Illinois M. J. **36**:5 (July) 1919.

Thyroglossal Duct, Cyst of. O. T. Freer.
Illinois M. J. **36**:12 (July) 1919.

The American Medical Association will pay thirty cents each for the following issues of the AMERICAN JOURNAL OF DISEASES OF CHILDREN: January, 1913; February, March, November and December, 1914; January and August, 1915; January, 1918; March and April, 1919. Address to AMERICAN MEDICAL ASSOCIATION, 535 North Dearborn Street, Chicago, Ill.